# The American Journal of Surgery

THURSTON SCOTT WELTON, M.D., *Editor*

*Associate Editors*
Bradley L. Coley, M.D., Arnold S. Jackson, M.D.,
E. Eric Larson, M.D.

PAPERS OF

## The American Association

FOR THE

## Surgery of Trauma

March 1953

SIXTY-SECOND YEAR OF PUBLICATION

EDITORIAL BOARD

# The American Journal of Surgery

*Editor* THURSTON SCOTT WELTON, *New York*

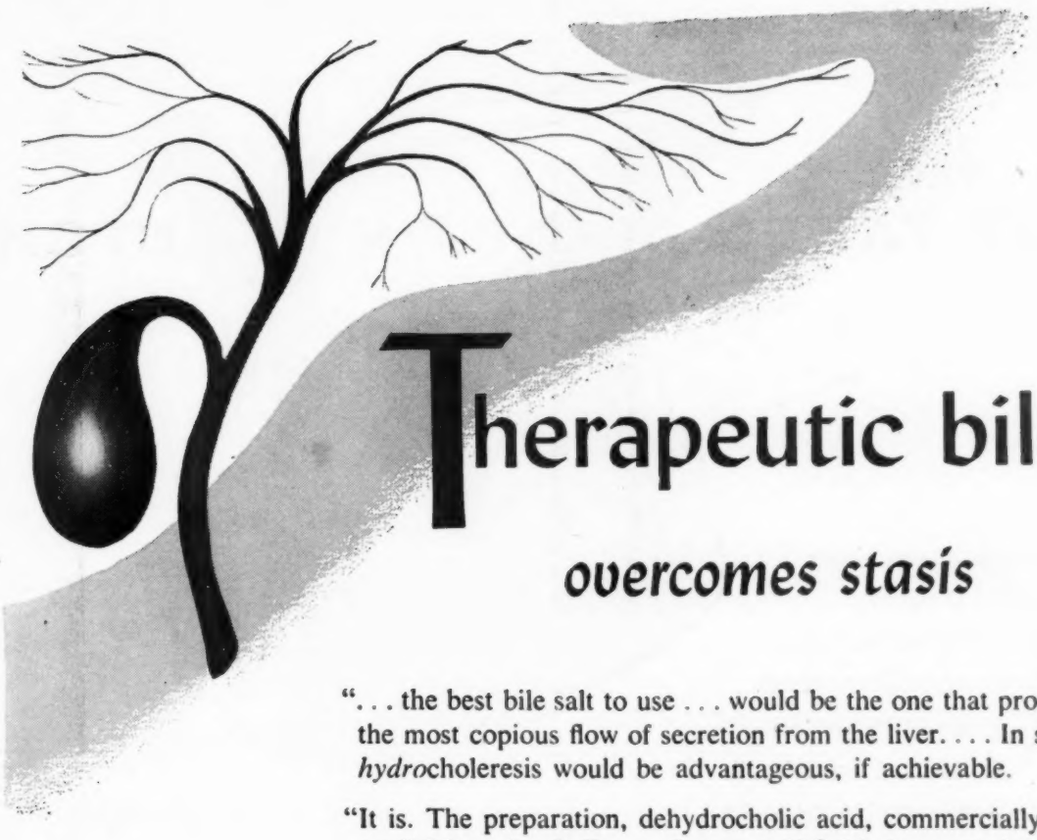
*Associate Editors* BRADLEY L. COLEY, *New York* · ARNOLD S. JACKSON, *Madison* · E. ERIC LARSON, *Los Angeles*

ADVISORY BOARD

CLAUDE S. BECK, *Cleveland*
CLARENCE J. BERNE, *Los Angeles*
GEORGE R. BRIGHTON, *New York*
MEREDITH F. CAMPBELL, *New York*
JAMES T. CASE, *Santa Barbara*
RANDOLPH LEE CLARK, JR., *Houston*
ISIDORE COHN, *New Orleans*
FREDERICK A. COLLER, *Ann Arbor*
PAUL C. COLONNA, *Philadelphia*
GORDON S. FAHRNI, *Vancouver*
HERBERT C. FETT, *Brooklyn*
EMIL GOETSCH, *Brooklyn*
CHARLES A. GORDON, *Brooklyn*
DONALD GUTHRIE, *Sayre, Pa.*
LOUIS J. HIRSCHMAN, *Detroit*
EMILE F. HOLMAN, *San Francisco*
CLAUDE J. HUNT, *Kansas City, Mo.*
THOMAS J. KIRWIN, *New York*
*New York*, ARTHUR KRIDA
*Chicago*, WALTER G. MADDOCK
*New Orleans*, URBAN MAES
*Newark*, HARRISON S. MARTLAND
*New Orleans*, RUDOLPH MATAS
*St. Louis*, CARL A. MOYER
*Toronto*, D. W. GORDON MURRAY
*San Francisco*, H. C. NAFFZIGER
*Baltimore*, EMIL NOVAK
*Newark*, CLARENCE R. O'CROWLEY
*Boston*, LOUIS E. PHANEUF
*Rochester, Minn.*, JAMES T. PRIESTLEY
*Raleigh*, HUBERT A. ROYSTER
*Philadelphia*, HENRY S. RUTH
*Memphis*, ROBERT L. SANDERS
*Baltimore*, GRANT E. WARD
*Woodland, Calif.*, J. HOMER WOOLSEY

*The American Journal of Surgery is published monthly, by The American Journal of Surgery, Inc., 49 West 45th Street, New York 36, N. Y. Yearly Subscription, $12.00 U. S. A.; $13.00 Canada and Pan-American Countries; $15.00 Foreign. Single Numbers $2.00; Special Numbers $4.00. Reentered as second class matter February 7, 1951, at the Post Office, New York, N. Y. and on February 7, 1951, at York, Pa., under the act of March 3, 1879. March 1953—New Series, Volume LXXXV, No. 3.* *Copyright, 1953, by The American Journal of Surgery, Inc.*

*Change of address must reach us one month preceding month of issue.*



# Therapeutic bile

## *overcomes stasis*

"... the best bile salt to use ... would be the one that produced the most copious flow of secretion from the liver. ... In short, *hydro*choleresis would be advantageous, if achievable.

"It is. The preparation, dehydrocholic acid, commercially available as Decholin ... does considerably increase the volume output of a bile of relatively high water content and low viscosity. The drug is not a cholagogue, i.e., it does not promote evacuation of the gallbladder, but it is a good 'flusher'."*

# Decholin®

**dehydrocholic acid, Ames**

*Hydro*choleresis with *Decholin* produces abundant, thin, free-flowing bile—"therapeutic bile." This flushes thickened bile, mucus plugs and debris from the biliary tract.

*Decholin* Tablets, 3¾ gr. (0.25 Gm.), bottles of 100, 500, 1000 and 5000.

*Decholin Sodium* (sodium dehydrocholate, *Ames*) 20% aqueous solution, ampuls of 3 cc., 5 cc., and 10 cc.

*Beckman, H.: Pharmacology in Clinical Practice, Philadelphia, W. B. Saunders Company, 1952, p. 361.

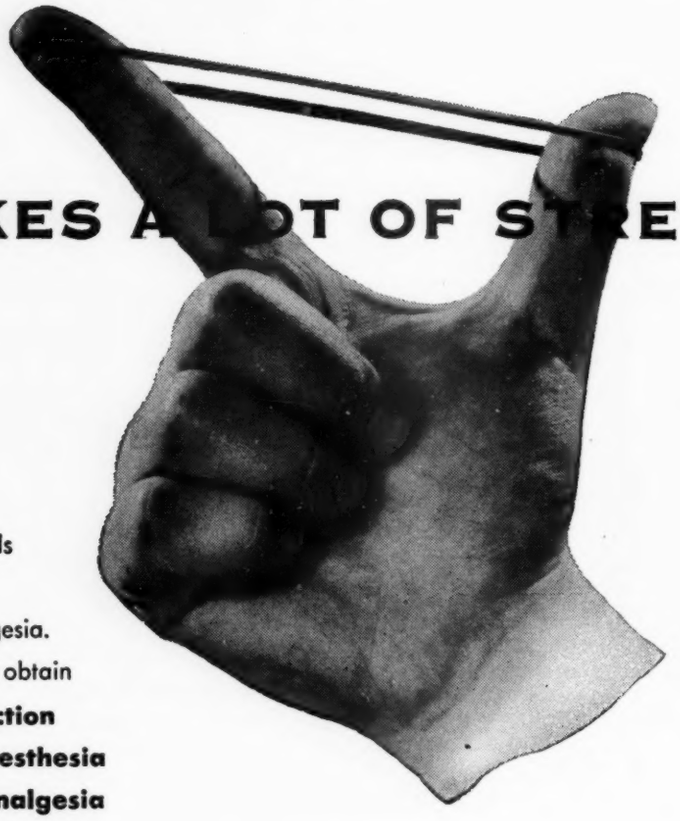
*Decholin* and *Decholin Sodium*, trademarks reg.

AMES

COMPANY, INC., ELKHART, INDIANA

Ames Company of Canada, Ltd., Toronto

46753



# IT TAKES A LOT OF STRETCH

. . . to cover all your needs
when it comes to
anesthesia and analgesia.
With Pontocaine you obtain

**prompt induction**
**sustained anesthesia**
**prolonged analgesia**
**relative safety and**
**convenience**

Surgeons and anesthesiologists who use Pontocaine for nerve block and infiltration analgesia know that you can stretch operating time to as much as five hours and postoperative analgesia to nine hours.

Pontocaine's much longer duration of action allows you to complete a surgical procedure without fear that the anesthesia may wear off . . . without apprehension that the postoperative analgesia won't be long enough to give your patient a good start towards recovery.

And it takes so little Pontocaine to do it . . . only 0.1 to 0.2 per cent concentration . . . leaving you with a relatively *wide margin of safety.*

**PONTOCAINE®**
HYDROCHLORIDE

Winthrop-Stearns Inc.
New York 18, N. Y. • Windsor, Ont.

sustained anesthesia
2 to 5 hour nerve block
2 to 3 hour spinal
prolonged analgesia
5 to 9 hour pain relief

**For 5 hour nerve block**
(surgical, diagnostic
and therapeutic),
for Infiltration and Continuous
Caudal Analgesia
0.15 per cent solution,
vials of 100 cc.

**For 2 to 3 hour spinal anesthesia**
"Niphanoid"® 10 mg.,
15 mg. and 20 mg.
1 per cent solution,
ampuls of 2 cc. (20 mg.)

Also available as
0.5 and 2 per cent solutions
for topical application.

Pontocaine (brand of tetracaine) and Niphanoid, trademarks reg. U.S. & Canada

# The American Journal of Surgery

VOL. LXXXV — Contents • March 1953 — NUMBER THREE


## Presidential Address


THE FUTURE OF THE TREATMENT OF TRAUMA . . . . . *Arthur R. Metz* 249


## Scientific Papers


FRACTURE OF THE NECK OF THE HUMERUS WITH DISLOCATION OF THE HEAD FRAGMENT . *Charles S. Neer, Thomas H. Brown, Jr. and Harrison L. McLaughlin* 252

PARTIAL CLAVICULECTOMY . . . . . *Carruth J. Wagner* 259

EPIPHYSEAL INJURIES OF THE RADIAL HEAD AND NECK. . . . . *Sawnie R. Gaston, Frederick M. Smith and Orren D. Baab* 266

REFRACTURES IN CHILDREN . . . *William H. Cassebaum and Richard B. Hamilton* 277

FRACTURES IN METASTATIC CARCINOMA . . . . . *William T. Fitts, Jr., Brooke Roberts and Isidore S. Ravdin* 282

COMMON PITFALLS AND COMPLICATIONS IN HIP NAILING . . . . . *F. P. Maibauer and W. H. Honor* 290

SIMULTANEOUS BILATERAL TRAUMATIC DISLOCATION OF THE HIP. . . . *Kellogg Speed* 292

TRAUMATIC DISLOCATION OF THE HIP . . . *Ralph K. Ghormley and Roger Sullivan* 298

SECONDARY TENORRHAPHIES AND TENDON GRAFTS IN INJURIES TO THE HAND . . . . *Joseph L. Posch* 306

NERVE GRAFTS INTO HANDS . . . . . *Henry C. Marble and Charles B. Burbank* 319


*Contents Continued on Page 5*

# Now available for civilian use!..

# *Gentran** (DEXTRAN) SOLUTION

## an effective, proven plasma volume expander for use in the treatment of shock

***Gentran*** is a clinically effective and safe plasma volume expander designed for the prevention and treatment of shock due to hemorrhage, burns, surgical procedures, and other conditions. Prepared for intravenous infusion, it is a sterile, nonpyrogenic 6% w/v solution of dextran in saline.

. . . does not interfere with typing or cross-matching . . . requires no refrigeration . . . is liquid at ordinary temperatures . . . available to your hospital . . . 500 cc. containers . . . easily administered with standard Plexitron solution set . . .

*trade name

*products of*

**BAXTER LABORATORIES, INC.**

Morton Grove, Illinois • Cleveland, Mississippi

DISTRIBUTED AND AVAILABLE ONLY IN THE 37 STATES EAST OF THE ROCKIES (except in the city of El Paso, Texas) THROUGH

AMERICAN HOSPITAL SUPPLY CORPORATION

GENERAL OFFICES • EVANSTON, ILLINOIS

# The American Journal of Surgery

Reg. U. S. Pat. Off., Nov. 3, 1936

VOL. LXXXV — Contents • March, 1953 — NUMBER THREE



MANAGEMENT OF SEVERE TRAUMA TO DISTAL DIGITAL EXTREMITIES. . . . . *George F. Crikelair, Robert T. Crowley and Harry Miller* 322

EPIDERMOID (EPITHELIAL) CYST OF THE HAND SKELETON . . . . . *Robert E. Carroll* 327

CONSERVATIVE MANAGEMENT OF LOW BACK PAIN. . . . . *B. E. McConville* 335

MANAGEMENT OF LOW BACK AND SCIATIC PAIN . . . . . *G. Edmund Haggart and William R. Grannis* 339

MALIGNANT SYNOVIOMA OF THE ANKLE. . . . . *Donald C. Conzett* 350

TRAUMA AND DUPUYTREN'S CONTRACTURE. . . . . *John J. Moorhead* 352

COMPARATIVE EXPERIMENTAL STUDIES ON THE TREATMENT OF TRAUMATIC SHOCK . . . *Charles L. Fox, Jr., Walter L. Mersheimer, Sigmund Lasker and James M. Winfield* 359

FAT EMBOLISM . . . *Capt. W. W. Glas, Capt. T. D. Grekin and M. M. Musselman* 363

TETANUS REPORT OF TWO FATAL CASES FOLLOWING TRAUMA . *Walter Scott Nettrour* 370

SELECTIVE PHYSIOLOGIC DÉBRIDEMENT WITH TOPICAL TRYPTIC ENZYMES . . . . . *Howard G. Reiser, L. C. Roettig and G. M. Curtis* 376

THE PLASMA SUBSTITUTES. PROGRESS REPORT . . . . . *Brig. Gen. Sam F. Seeley and Lieut. Col. Edwin J. Pulaski* 382

A MIDDLE COURSE IN BLOOD AND FLUID REPLACEMENT IN TRAUMA. . *John Paul North* 386

ILIAC BONE GRAFTS REPLACING TANTALUM PLATES FOR GUNSHOT WOUNDS OF THE SKULL. *Clifford L. Kiehn and Antonio Grino* 395

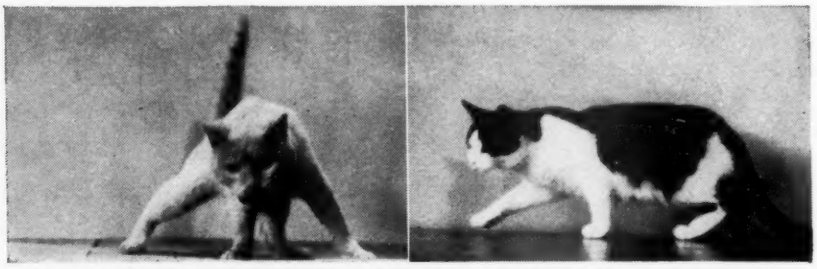

*Contents Continued on Page 7*

# New principle

## in

# Streptomycin Therapy

The hazard of ototoxicity is greatly reduced by combining equal parts of streptomycin sulfate and dihydrostreptomycin sulfate. The patient thus gets only *half* as much of each drug. The risk of vestibular damage (from streptomycin) and of hearing loss (from dihydrostreptomycin) is greatly reduced. Therapeutic effect is undiminished. This principle has been demonstrated in both animals and man. In patients treated for 120 days with 1 Gm. per day of the combined drugs, the incidence of neurotoxicity was practically zero.



Cat treated with streptomycin is ataxic.

Cat treated with the same amount of streptomycin-dihydrostreptomycin has normal equilibrium.

**DISTRYCIN**
(di-STRI-sin)

Squibb Streptomycin Sulfate *and* Dihydrostreptomycin Sulfate in equal parts

**For greater safety in combined antibiotic therapy**
These new formulations embody this new principle:

| | DICRYSTICIN | DICRYSTICIN FORTIS | DISTRYCILLIN A. S. |
|---|---|---|---|
| Procaine penicillin G, units | 300,000 | 300,000 | 400,000 |
| Potassium penicillin G, units | 100,000 | 100,000 | — |
| Streptomycin sulfate, Gm. | 0.25 | 0.5 | 0.25 |
| Dihydrostreptomycin sulfate, Gm. | 0.25 | 0.5 | 0.25 |

(All supplied in 1 and 5 dose vials)

*'Distrycin' and 'Dicrysticin' are registered trademarks; 'Distrycillin' is a trademark.*

SQUIBB
*a leader in the research and manufacture of penicillin and streptomycin*

# The American Journal of Surgery

Reg. U. S. Pat. Off., Nov. 3, 1936

VOL. LXXXV — Contents • March 1953 — NUMBER THREE



BONE GRAFTS FOR LARGE GAPS IN MANDIBLE . *James Barrett Brown and Minot P. Fryer* 401

ACCELERATED EXCISION AND GRAFTING OF SMALL DEEP BURNS . . . *Frank McDowell* 407

TREATMENT OF BURNS BY EXCISION AND IMMEDIATE SKIN GRAFTING . *Alfred H. Whittaker* 411

RUPTURE OF BLOOD VESSELS BY STRENUOUS PHYSICAL EXERTION . . . . . *Chester C. Guy and W. A. Wichowski* 418

ARTERIAL REPLACEMENT IN TRAUMA . . . . . *Prescott Jordan, Jr., Tor Hiertonn and Charles G. Johnston* 424

RELATION OF TRAUMA TO LESIONS OF THE MALE BREAST . . . . . *W. Wallace Greene and Nelson J. Howard* 431

SHOTGUN WOUNDS. . . . . *James C. Drye and George Schuster* 438

## *Symposium on Skeletal Trauma*

LOCAL PHYSIOLOGY OF BONE REPAIR. WITH PARTICULAR REFERENCE TO THE PROCESS OF NEW BONE FORMATION BY INDUCTION . . *Marshall R. Urist and Franklin C. McLean* 444

VASCULAR COMPLICATIONS AND SEQUELAE OF SKELETAL TRAUMA . *Louis G. Herrmann* 450

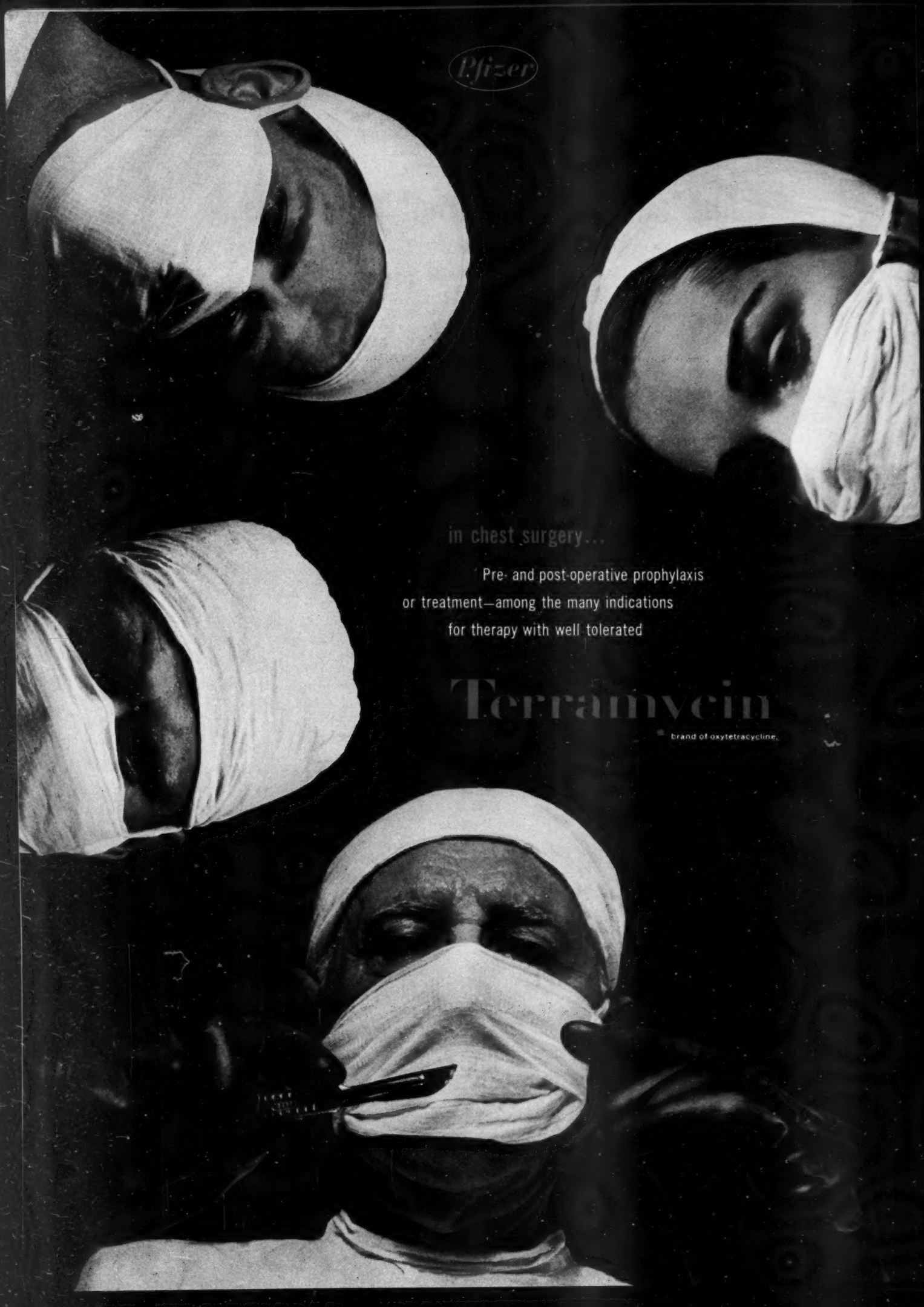
COMPLICATIONS AND SEQUELAE OF INFECTION IN SKELETAL TRAUMA . . . . . *Oscar P. Hampton, Jr.* 455

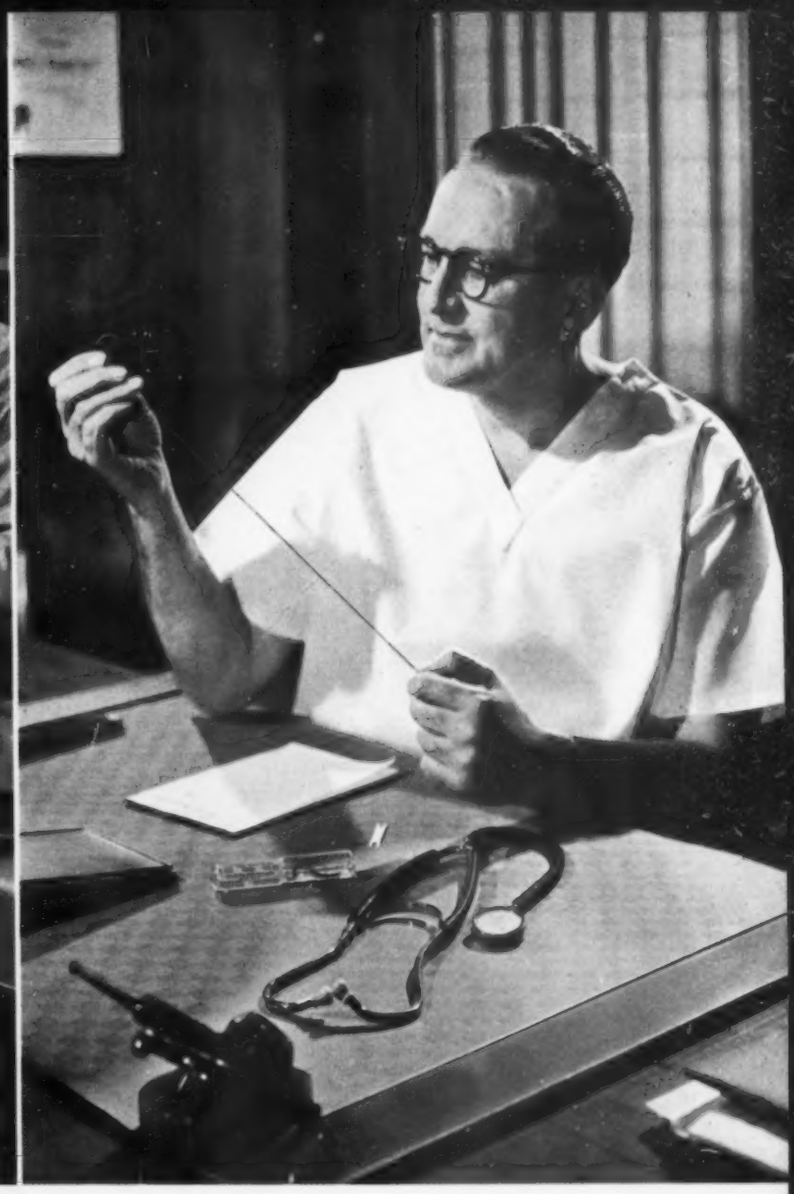
PRIORITY EVALUATION AND MANAGEMENT OF MULTIPLE INJURIES . . . *Frank Glenn* 461

OFFICERS OF THE AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA, 1952–1953. . 468


*Advertising Index on 3rd Cover*

*Change of address must reach us one month preceding month of issue.*


Pfizer
in chest surgery...
Pre- and post-operative prophylaxis
or treatment—among the many indications
for therapy with well tolerated
Terramycin
brand of oxytetracycline.



# Why not choose a suture as carefully as a pair of shoes?

Strangely enough, it makes good sense to talk about sutures and shoes on the same page!

Proper tanning of leather is vital to shoe quality. And quality in catgut sutures depends on a process much like tanning — the chromicizing process.

If the hide isn't tanned all the way through, an inferior grade of leather will result. Likewise, a suture not thoroughly chromicized will often have a surface that resists absorption and a core that absorbs too fast.

Tanners recognize the advantages of a *two-bath* tanning process for fine leather. A similarly thorough process is the *new* Curity *two-bath method of chromicizing* sutures. Only total, even chromicization can give you dependable absorption performance in a suture. For further dependability, Curity sutures are chromicized only *after* catgut plies have been firmly bonded into strands by natural gut mucin. This method requires no foreign bonding agents.

The modern Curity Chromic Suture is another better tool of surgery—from the laboratory which has made major contributions to suture making.

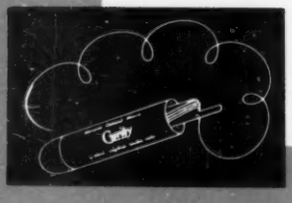
Curity
REG. U.S. PAT. OFF.
SUTURES

BAUER & BLACK
DIVISION OF THE KENDALL COMPANY
309 W. Jackson Blvd., Chicago 6

# Curity chromic sutures

REG. U.S. PAT. OFF.

## are chromicized with two baths for dependable absorption



**Better control of suture absorption rate is now possible with the new Curity method of two-bath chromicizing after the strand has been formed. The first bath *does not* "*tan*" but permeates the strand. The solution used in the second bath combines with the molecules of the first, within the strand, achieving total, even chromicization from rim to center. As a result absorption is similarly uniform. By this method the plies are bonded by their natural mucin.**

*Reasons for effectiveness of Furacin*

A wide antibacterial spectrum, including many gram-negative and gram-positive organisms • Effectiveness in the presence of wound exudates • Lack of cytotoxicity: no interference with healing or phagocytosis • Low incidence of sensitization: less than 5% • Ability to minimize malodor of infected lesions • Stability.

Furacin® preparations contain Furacin 0.2% brand of nitrofurazone N.N.R. in water-miscible vehicles which dissolve in exudates.

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

FURACIN"
SOLUBLE DRESSING
(BRAND OF NITROFURAZONE)
N. N. R.
CONTAINS 0.2% NITROFURAZONE N. N. R. IN A WATER SOLUBLE BASE
PRESCRIPTION OF A PHYSICIAN.
CAUTION: TO BE DISPENSED ONLY BY OR ON THE
WARNING: THIS DRUG MAY CAUSE SENSITIZATION. IF THIS OCCURS, ITS USE SHOULD BE DISCONTINUED.
LITERATURE AVAILABLE TO PHYSICIANS ON REQUEST
AN ANTIBACTERIAL PREPARATION FOR TOPICAL APPLICATION
EATON Inc.
NORWICH, NEW YORK
ONE POUND, AVOIR.
453.6 GRAM

**IN MALODOROUS LESIONS...**

The effective antibacterial action of Furacin can rapidly abate malodor. Such benefit has been reported in a variety of conditions: diabetic gangrene, varicose ulcers, chronic wounds, malignant lesions, otitis media.*

*Downing, J. G. et al.: J.A.M.A. 133:299 1947. Shipley, E. R. et al.: Surg. Gynec. & Obst. 84:366 1947. Wawro, N. W.: Connecticut M. J. 12:17 1948. McCollough, N. C.: Indust. Med. 16:128 1947. • Long, P. H.: A-B-C's of Sulfonamide and Antibiotic Therapy, Philadelphia, W. B. Saunders, 1948, p. 152. • Meyer, J. H.: J. Internat. Coll. Surg. 13:748 1950.

*Literature on request*

EATON LABORATORIES Inc.
NORWICH, NEW YORK

The NITROFURANS
A unique class of antimicrobials

**FURACIN SOLUBLE DRESSING • FURACIN SOLUTION • FURACIN ANHYDROUS EAR SOLUTION**

# B-P RIB-BACK
## *is more than a name*

It is the hallmark of a fine surgical blade by any standard, reflecting infinite capacity for attention to every detail of quality production.

This means . . . uniform sharpness resulting in maximum cutting performance for the surgeon . . . reduction of time-consuming delays for the entire surgical team . . . an investment in economy for the budget-wise P. A.

B-P blades are now wrapped without oil in a chemical, rust-inhibiting package. No wiping is necessary before sterilization. Unused blades in an opened package may be rewrapped—still protected against corrosion.

*Ask your dealer*

**BARD-PARKER COMPANY, INC.**
**Danbury, Connecticut**

# WAPPLER
## *Cold Cautery*
# SCALPEL

**A Powerful**
**High Frequency Apparatus for**
**Electro Surgical Procedures**

*Note*
*these Convenience Features*

THE WAPPLER COLD CAUTERY SCALPEL is a miniature high frequency apparatus which provides a high frequency current that can be utilized both for cutting and coagulation. The name "Cold Cautery" was selected to describe the effect of the current when applied to tissue because the electrode itself is cold, while the hot point, or thermal cautery, severs tissue by a searing contact of a heated metal wire electrode. The thermal or hot point cautery has certain limitations for operative work, such as excessive tissue destruction by radiation of heat, which in turn creates severe scar formation.

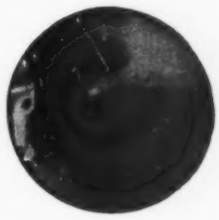
The Wappler Cold Cautery Scalpel being a high frequency apparatus, provides cutting and coagulating current which may be precisely controlled in intensity.

**SPECIFICATIONS**

**Model C-450** is for operation on 110V. A.C. 50-60 cycles.
**Model C-450A** is for operation on 220V. A.C. 50-60 cycles.
**Model C-450B** is for operation on 110V. A.C. 25 cycles.
*A footswitch and vacuum tubes are furnished with each model.*

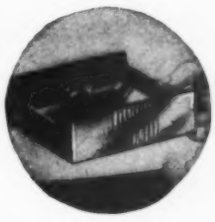
WAPPLER A·C·M·I TRADE MARK

**WRITE FOR LITERATURE**
*which completely describes various sets of electrodes and accessory instruments.*



Conducting cords leading to the active and indifferent electrodes are housed within the case. To return the cords to their original position, pressure on the button above them instantly rewinds the cord.

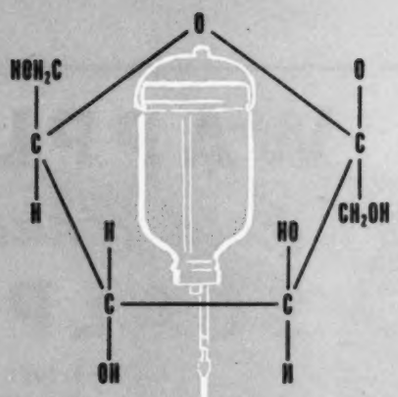
The drawer and contents are automatically ejected from the case upon pressure on a button located directly above the drawer.



The cord connector of the shock-proof footswitch is plugged into the outlet marked "Footswitch." Bipolar electrodes, handle and cord are plugged into the receptacle marked "Bipolar."

Two outlets marked "Light" in center of the panel provide a means of supplying diagnostic light source. Conducting cords may be immersed in sterilizing solution in tray.

**AMERICAN CYSTOSCOPE MAKERS, INC.**
1241 LAFAYETTE AVENUE, NEW YORK 59, N. Y.



presenting

# Levugen

(FRUCTOSE, MEAD)

## fructose (levulose) for intravenous infusion . . . now, for the first time, available for general clinical use

The advantages of fructose for intravenous infusion have long been recognized.[1,2] But limited availability of pure fructose has prevented its general clinical use.

Now, extensive research in carbohydrate chemistry by Mead Johnson & Company has resulted in a practical and economical method of producing pure fructose. As Levugen 10% in Water, it is available for intravenous use.

"Levugen *can be infused more rapidly*"

# Levugen (Fructose, Mead) can be infused much more rapidly than dextrose, with better retention and less disturbance of fluid balance

Since Levugen is removed from the blood very rapidly, it does not produce high hyperglycemic levels or spill over into the urine in significant amounts even when it is infused in fairly high concentration.[1,2,3] Levugen can therefore be given much more rapidly than dextrose, with less loss of potential calories through glycosuria. A liter of Levugen 10% in Water can be given in the same time as a liter of 5% dextrose.

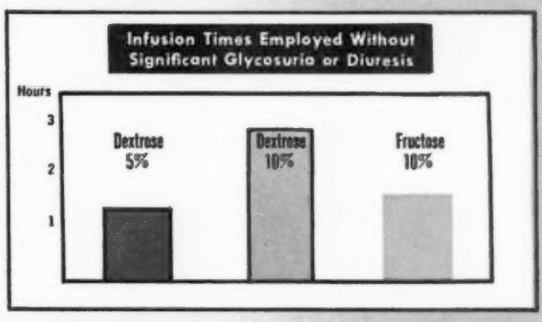
Experimental evidence indicates, moreover, that Levugen is more rapidly and completely utilized for hepatic glycogen formation than is dextrose.[1,4]

## Clinical Advantages of Levugen

- More rapid and adequate provision of calories
- Less loss of calories through glycosuria
- More rapid formation of liver glycogen
- Less disturbance of fluid balance
- Shorter infusion time, with less discomfort for the patient
- Less time and trouble for hospital personnel

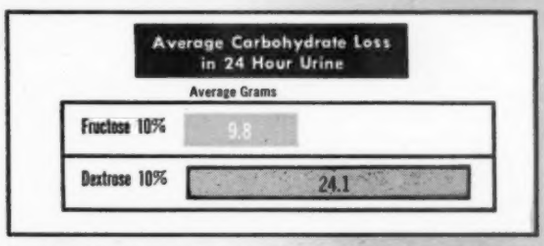
Levugen 10% in Water is available in 1 liter (1000 cc.) flasks, containing 100 Gm. of Levugen (Fructose, Mead), approximately 400 calories.

**LEVUGEN GIVEN TWICE AS RAPIDLY AS DEXTROSE**




Based on infusion rates per liter reported by Lockhart and Elman[3] (for dextrose) and Weichselbaum et al.[2] (for fructose).

**MUCH LESS URINARY LOSS OF CALORIES WITH LEVUGEN**




Based on data from Elman.[5] Infusion of 2 liters of each solution into the same subjects on alternate days.

*References:* (1) Nutrition Rev. 9: 173-175, 1951; (2) Weichselbaum, T. E., et al.: Proc. Soc. Exper. Biol. & Med. 75: 816-818, 1950; (3) Lockhart, C. E., and Elman, R.: Surg., Gynec. & Obst. 88: 97-102, 1949; (4) Cori, C. F.: J. Biol. Chem. 70: 577-588, 1926; (5) Elman, R.: Personal Communication.

**Levugen**

(FRUCTOSE, MEAD)

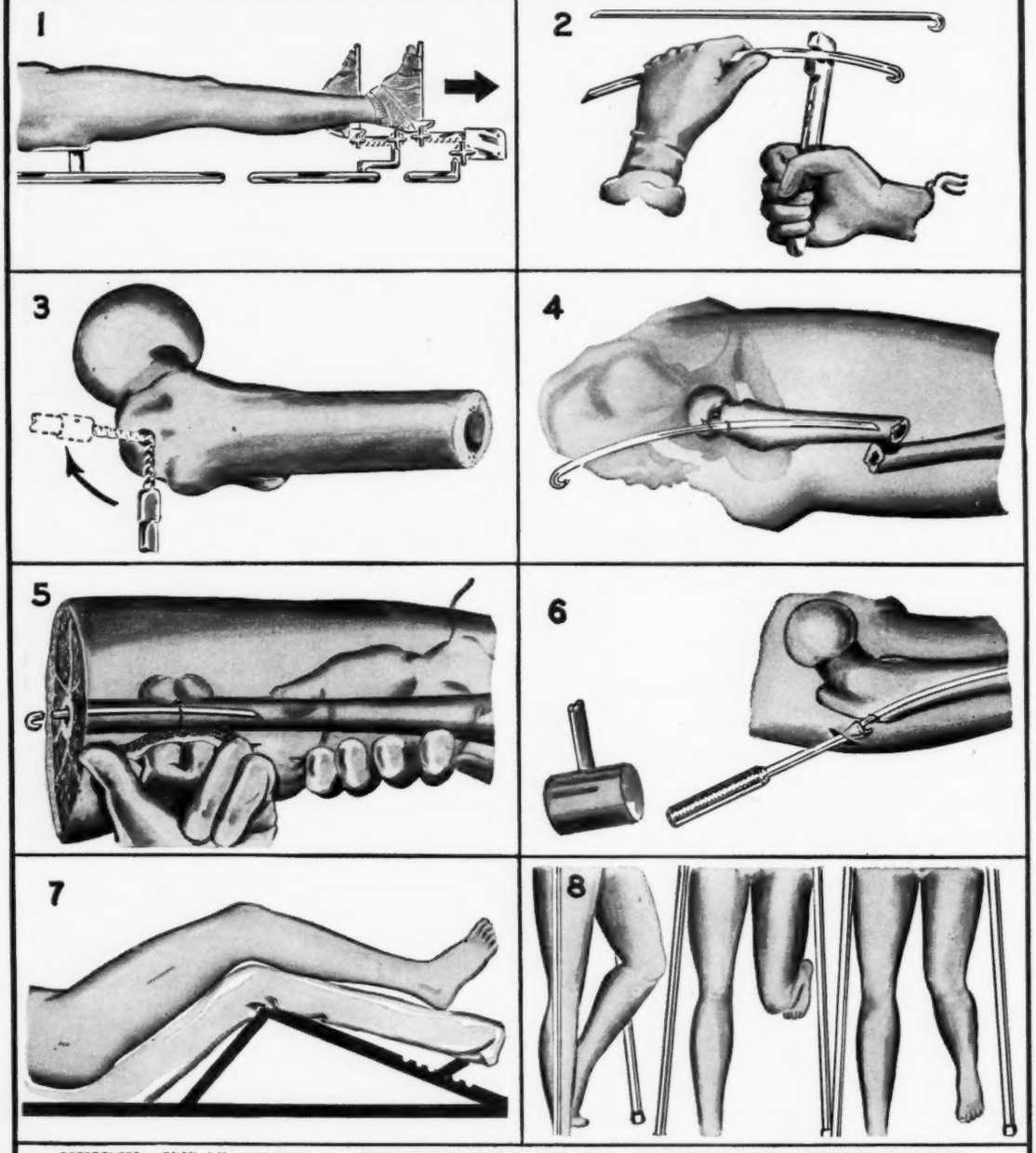
MEAD

MEAD JOHNSON & COMPANY • EVANSVILLE 21, IND., U.S.A.

# The RUSH MEDULLARY PIN

PATENTED

# PIN-A-GRAM - FEMUR -




REFERENCES: RUSH, L.V.; AND RUSH, H.L., *THE MISSISSIPPI DOCTOR*, SEPT., 1949; *THE AMERICAN JOURNAL OF SURGERY*, SEPT., 1949

SEMI-OPEN REDUCTION
LESS TRAUMA
DISSECTION OF BONE UNNECESSARY

WRITE FOR INFORMATION

MFG. EXCLUSIVELY BY:

THE BERIVON Company

P.O. BOX 1851
Meridian, Miss.

for maximum safe analgesia . . .

# Phenaphen®

## with Codeine

¼ GR. OR ½ GR.

Provides full codeine effect with small, safe codeine dosage — by synergistic combination with the potent, non-narcotic Phenaphen formula*.

A complete analgesic for control of pain, and of the patient's emotional reaction to pain.

***Three forms:***

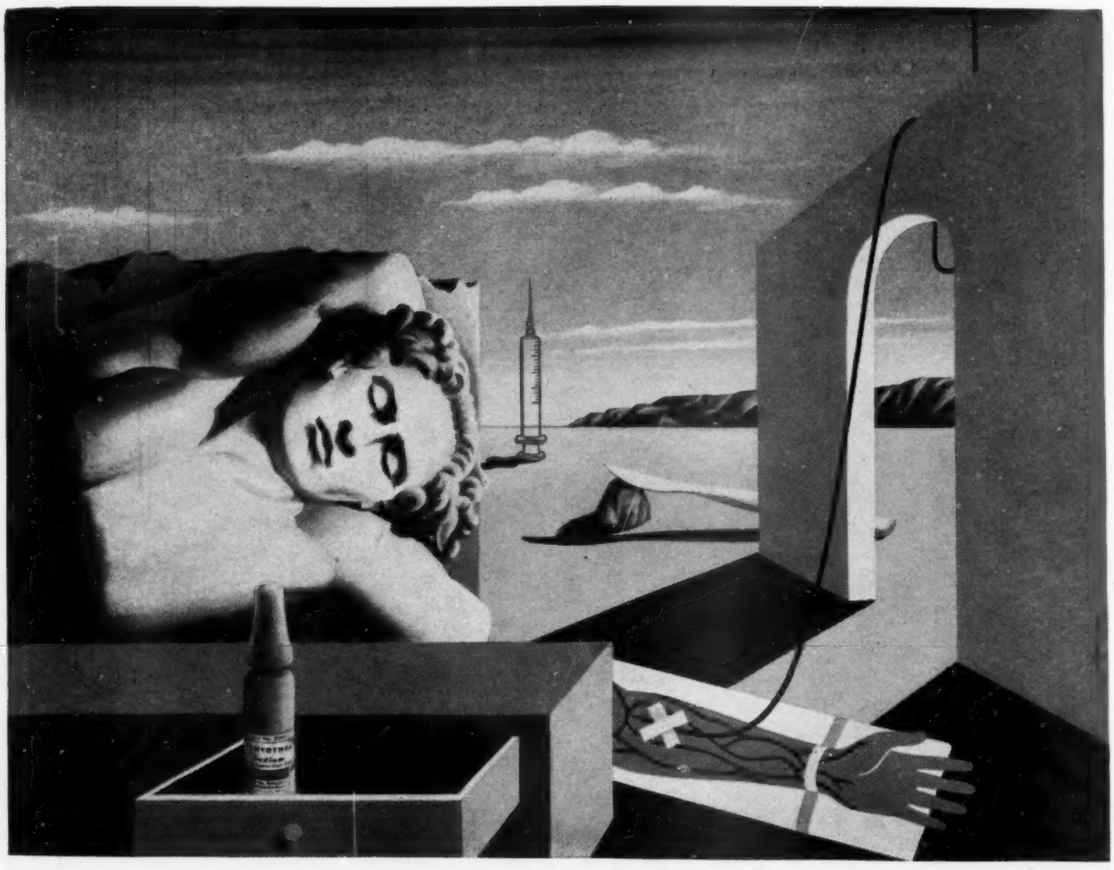
**PHENAPHEN**
— the basic non-narcotic preparation
(brown and white capsules)

**PHENAPHEN WITH CODEINE PHOSPHATE ¼ GR.**
— *PHENAPHEN No. 2*
(yellow and black capsules)

**PHENAPHEN WITH CODEINE PHOSPHATE ½ GR.**
— *PHENAPHEN No. 3*
(green and black capsules)

**A. H. ROBINS CO., INC.**
*Ethical Pharmaceuticals of Merit since 1878*
**RICHMOND 20, VIRGINIA**

**Each Phenaphen Capsule contains aspirin (2½ gr.) 162 mg., phenacetin (3 gr.) 194 mg., hyoscyamine sulfate 0.031 mg., and phenobarbital (¼ gr.) 16.2 mg.*



**"Actually, it has not been so much a case of PENTOTHAL Sodium's supplanting other anesthetic agents and methods as it has been of complementing and supplementing them to the mutual advantage of one another."**

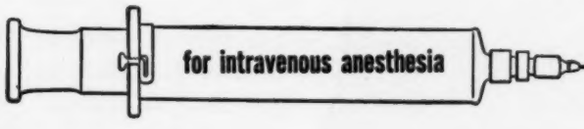
Adams, R. Charles (1951), Intravenous Administration of Pentothal Sodium in Combination with Other Anesthetic Agents and Methods, J. Missouri Med. Assn., August.

1-105

IN minor and major surgery, for induction or induction and maintenance, *alone or in combination* with other anesthetics, PENTOTHAL Sodium continues to grow in popularity in operating rooms throughout the world.

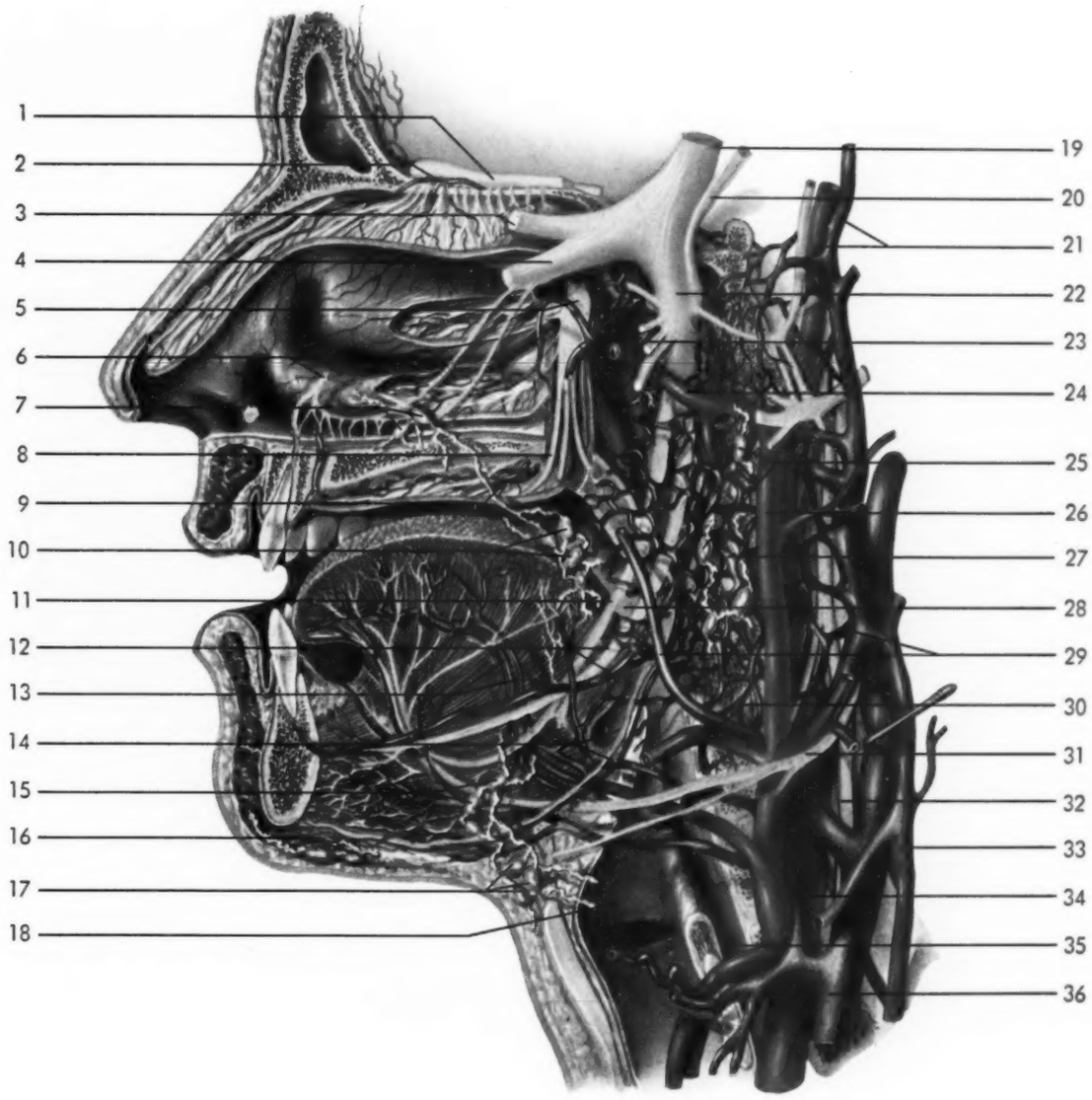
Nineteen years of experience, nearly 2000 published reports have shown that intravenous anesthesia with PENTOTHAL means a smooth, easy induction, and that deeper anesthesia may be had in a moment. Recovery is short, pleasant and usually without nausea. No bulky frightening equipment is needed. The fire and explosion hazard is eliminated. And, as it says above, this ultra-short-acting barbiturate complements and supplements other agents to "the mutual advantage of one another."

Abbott




PENTOTHAL® Sodium
(STERILE THIOPENTAL SODIUM, ABBOTT)

# Anatomy *of the* Mouth




1. Olfactory nerve
2. Anterior ethmoidal artery
3. Ophthalmic nerve
4. Maxillary nerve
5. Sphenopalatine ganglion
6. Anterior, middle & posterior superior alveolar nerves
7. Maxillary lymph nodes
8. Anterior palatine nerve
9. Great palatine artery
10. Buccinator lymph nodes
11. Lingual nerve
12. Inferior alveolar nerve & artery
13. Lingual artery & vein
14. Mylohyoid nerve & artery
15. Supramandibular lymph nodes
16. Submental lymph nodes
17. Submaxillary lymph nodes
18. Trachea
19. Sensory root of trigeminal nerve
20. Motor root of trigeminal nerve
21. Superficial temporal artery & vein
22. Mandibular nerve
23. Sphenopalatine artery
24. Internal maxillary artery
25. Parotid lymph nodes
26. External carotid artery
27. Pterygoid venous plexus
28. Oropharynx
29. Anterior & posterior facial veins
30. External maxillary artery
31. Hypoglossal nerve
32. Vagus nerve
33. External jugular vein
34. Internal carotid artery
35. Esophagus
36. Internal jugular vein

*This is one of a series of paintings by Paul Peck, illustrating the anatomy of various organs and tissues of the body which are frequently attacked by infection, where aureomycin may prove useful.*

Lederle

*When infections in the mouth are serious, frequently they will respond to*

# Aureomycin

HYDROCHLORIDE CRYSTALLINE

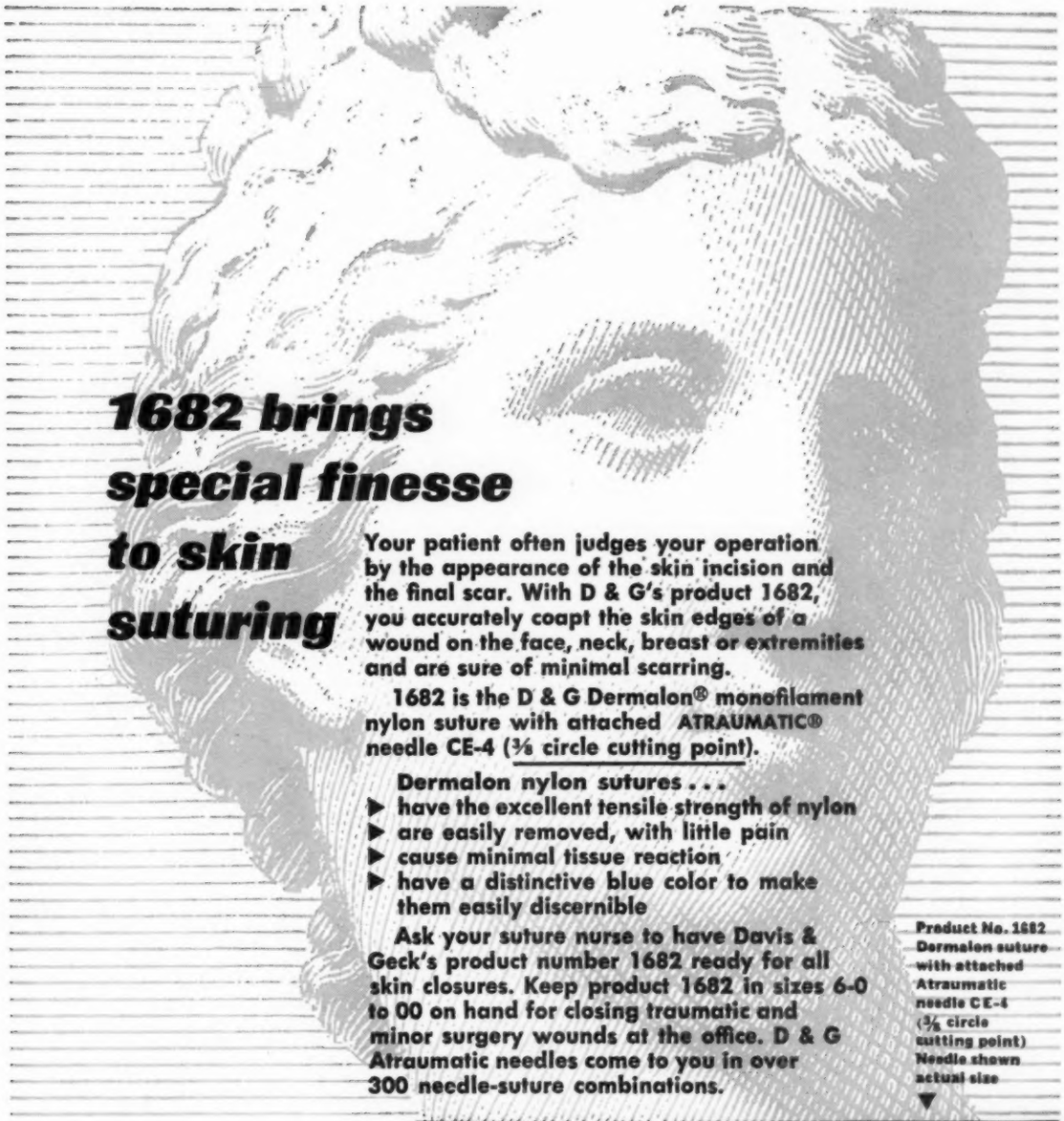
*Literature available on request*

* * *

LEDERLE LABORATORIES DIVISION

AMERICAN Cyanamid COMPANY

30 ROCKEFELLER PLAZA, NEW YORK 20, N.Y.



# 1682 brings special finesse to skin suturing

Your patient often judges your operation by the appearance of the skin incision and the final scar. With D & G's product 1682, you accurately coapt the skin edges of a wound on the face, neck, breast or extremities and are sure of minimal scarring.

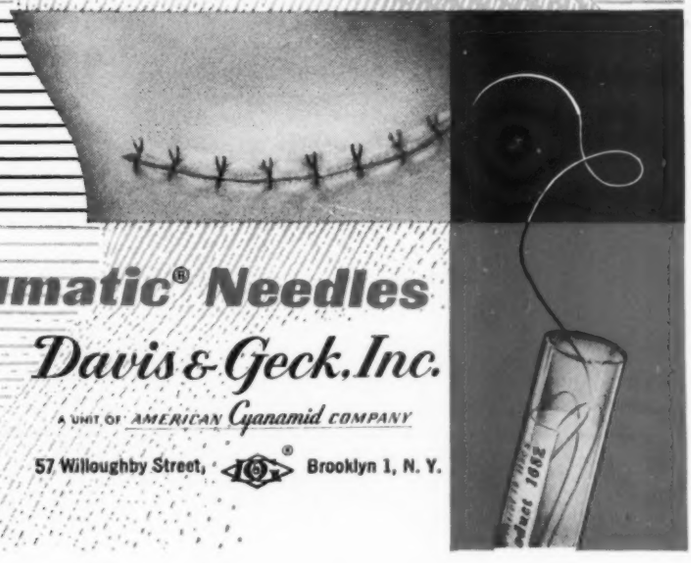
1682 is the D & G Dermalon® monofilament nylon suture with attached ATRAUMATIC® needle CE-4 (⅜ circle cutting point).

**Dermalon nylon sutures . . .**

- ▶ have the excellent tensile strength of nylon
- ▶ are easily removed, with little pain
- ▶ cause minimal tissue reaction
- ▶ have a distinctive blue color to make them easily discernible

Ask your suture nurse to have Davis & Geck's product number 1682 ready for all skin closures. Keep product 1682 in sizes 6-0 to 00 on hand for closing traumatic and minor surgery wounds at the office. D & G Atraumatic needles come to you in over 300 needle-suture combinations.

Product No. 1682 Dermalon suture with attached Atraumatic needle CE-4 (⅜ circle cutting point) Needle shown actual size ▼



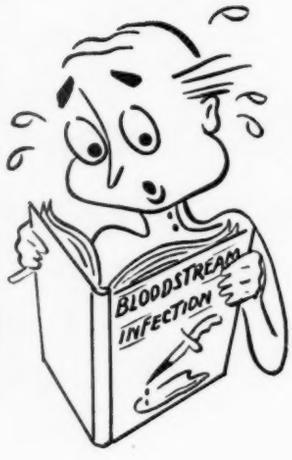
## Atraumatic® Needles

*Davis & Geck, Inc.*

A UNIT OF AMERICAN *Cyanamid* COMPANY

57 Willoughby Street, Brooklyn 1, N. Y.

# Killer Thriller




The literature* on bloodstream infection is eye-popping. From office injections to exodontia, *autocolave sterilization is a necessity* when you enter the bloodstream.

Boiling only nabs the easy ones, simple bacteria. The Castle "777" Speed-Clave kills *all* microbial life—even spore-bearers and viruses—gives your patients complete protection.

"777" is fast. Reaches sterilization heat in 7 minutes from a cold start (3 minutes if warm!). It's easy. Set it—that's all. No valves, no watching. Shuts off automatically. Saves current, saves instruments (less rust and dulling), saves 40 % on dressing costs—you can sterilize them dry, ready to use.

And, costs no more than a cabinet boiler. Call your Castle dealer for a painless demonstration, or write Wilmot Castle Co., 1149 University Ave., Rochester 7, N. Y.

* *We'll be glad to send you reprints*

*For a good laugh and a new look at sterilization—read our booklet, "The Untimely Ending of Jose Bacillus." Write for it, or ask your Castle dealer.*

**Castle LIGHTS and STERILIZERS**

# a new antibiotic

discovered in the Research Laboratories of Chas. Pfizer & Co., Inc. . . . clinically active *particularly* against penicillin–resistant gram-positive pathogens . . .

*particularly effective against*

# Magna

Experimental and clinical studies
indicate Magnamycin is
exceptionally well tolerated

---

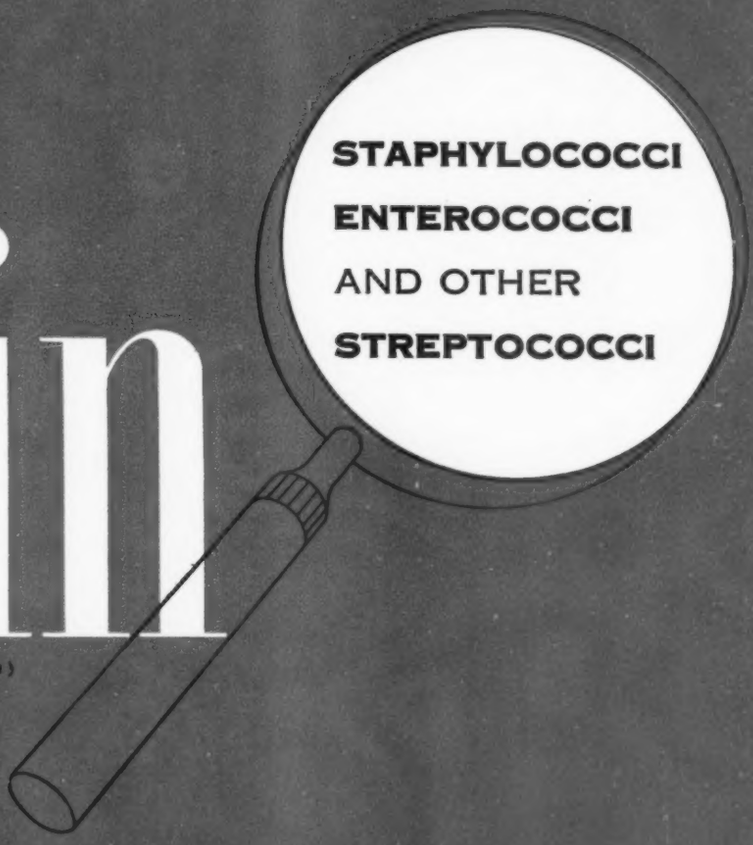
*Now available*

**Magnamycin Sugar Coated Tablets (100 mg.)**

**Bottles of 25 and 100**

ANTIBIOTIC DIVISION Pfizer

*those gram-positive organisms often resistant to penicillin*




# mycin

(BRAND OF CARBOMYCIN)

...active *in vitro* against other micro-organisms including:

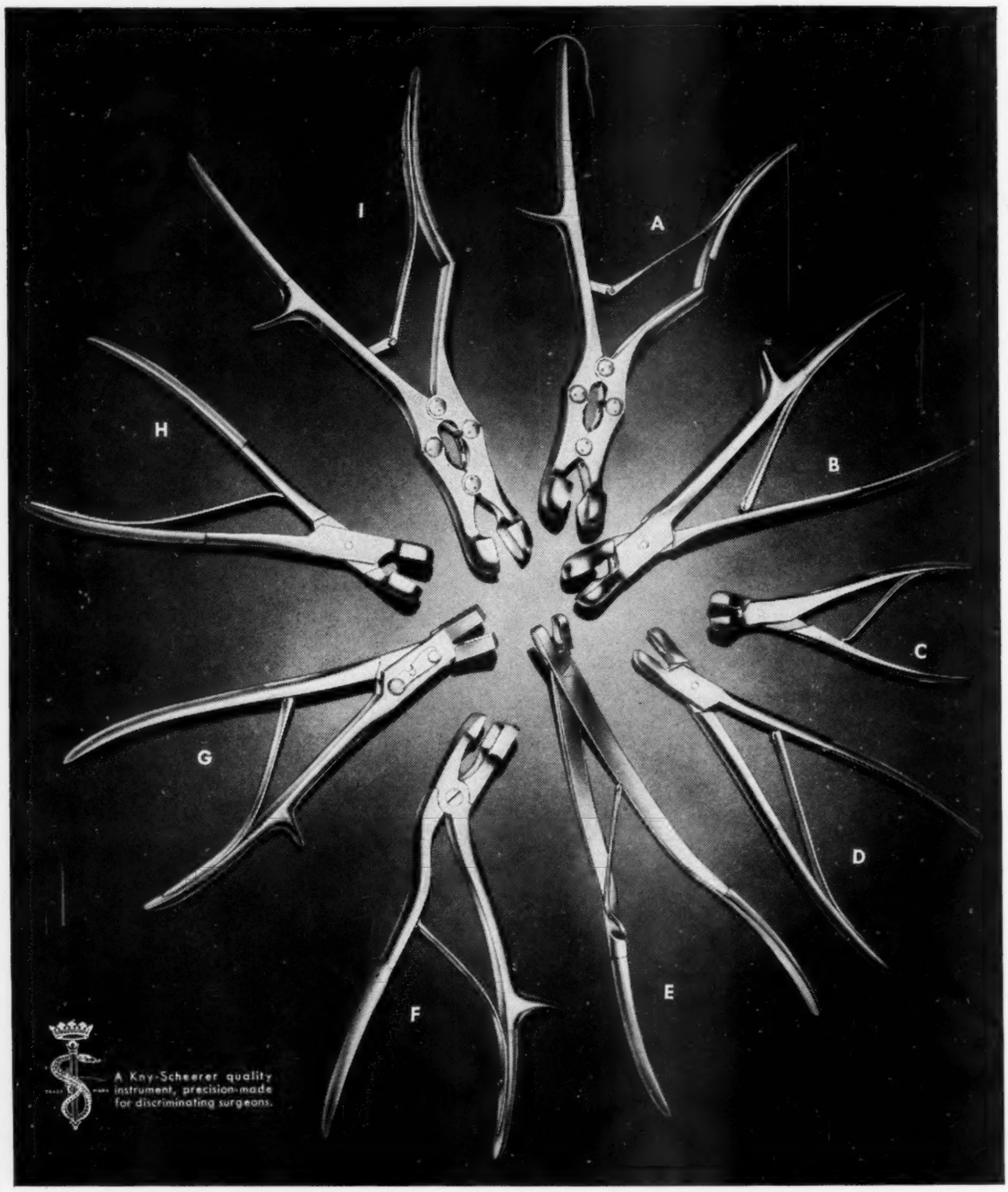
GONOCOCCI. PNEUMOCOCCI. MENINGOCOCCI. HEMOPHILUS INFLUENZAE

PLEUROPNEUMONIA-LIKE ORGANISMS

CORYNEBACTERIA. ERYSIPELOTHRIX. LISTERIA

CERTAIN RICKETTSIAL, VIRAL AND PROTOZOAN ORGANISMS

CHAS. PFIZER & CO., INC., *Brooklyn 6, New York*




**for rib resection . . .** when you want a rongeur, you want a *particular* rongeur. And you will find it in the KNY-SCHEERER line of finest quality, precision-made thoracic instruments, available exclusively through surgical dealers.

Consult your dealer for the *particular* KNY-SCHEERER rongeur that best suits your requirements . . . he will have it in stock or can quickly obtain it for you.

| | | |
|---|---|---|
| A | 2347 | Sauerbruch's Rib Rongeur, rounded jaws, 20 mm. wide, curved |
| B | 2342 | Sauerbruch's Rib Rongeur, curved, 20 mm. jaws, 12¼" long |
| C | 2343 | Sauerbruch's Rib Rongeur, square jaws, 15 mm. wide, 7¾" long |
| D | 2341½ | Sauerbruch's Rib Rongeur, curved, 9 mm. jaws, 11" long |
| E | 2344 | Lebsche's Rib Rongeur, for cutting stumps of first rib |
| F | 2348 | Lebsche's Sternum Punch, for exposing the pericardium |
| G | 2344½ | Rib Cutting Rongeur, for cutting stumps of first rib, square jaws, 10½" long |
| H | 2343½ | Sauerbruch's Rib Rongeur, square jaws, 20 mm. wide, 8¾" long |
| I | 2346½ | Sauerbruch's Rib Rongeur, rounded jaws, 20 mm. wide, straight |

**Kny-Scheerer** CORPORATION • 35 East 17th Street • New York 3, N. Y.

# clinically tested

# proved

# Efocaine*

## long-acting local anesthetic-analgesic

Clinical experience with thousands of patients and numerous journal reports testify to the fact that EFOCAINE goes far toward answering the long-felt medical and surgical need for prolonged non-narcotic pain control.

EFOCAINE, a depot solution, is supplied in 20-cc. multiple-dose vials. Technique of injection and dosages are fully described in package enclosure.

*Write for reprints and literature.*


**bibliography:**

Ansbro, F. P., and others: **Anesthesiology** *13*:306, 1952.

Ansbro, F. P., and others: **Journal Lancet**, in press.

Bartlett, R. W., and Eastwood, D. W.: **Surgery** *32*:956, 1952.

Bonica, J. J.: **J.A.M.A.** *150*:1581 (Dec. 20) 1952.

Borre, L. A.: **Arch. chir. néerl.**, in press.

Brown, J. M.: **M. Times, New York** *80*:583, 1952.

Cappe, B. E., and Pallin, I. M.: **Am. Pract. & Digest Treat.** *3*:739, 1952.

Crisp, W. E., and McDonald, R.: **Obstetrics and Gynecology**, in press.

Deaton, W. R., Jr., and Bradshaw, H. H.: **Am. Surgeon** *18*:616, 1952.

Bowlen, L. W.: **Bull. Dade County M. A.** *23*:30, 1952.

Gross, J. M., and Shaftel, H. E.: **New York State J. Med.** *52*:1413 (June 1) 1952.

Henderson, A. R.: **J. M. Soc. New Jersey** *49*:287, 1952.

Iason, A. H., and Shaftel, H. E.: **Am. J. Surg.** *83*:549, 1952.

Iason, A. H., and Shaftel, H. E.: **J. Internat. Coll. Surgeons**, in press.

Nylander, P. E. A.: **Ann. chir. et gynaec. Fenniae** *41*:1, 1952.

Penn, S. E.: **A.M.A. Arch. Otolaryng.** *56*:59, 1952.

Perrin, S. R.: **A.M.A. Arch. Dermat. & Syph.**, in press.

Puderbach, W. J., and Shaftel, H. E.: **Journal Lancet** 72:203, 1952.

Raicus, E.: **M. Times, New York** *80*:156, 1952.

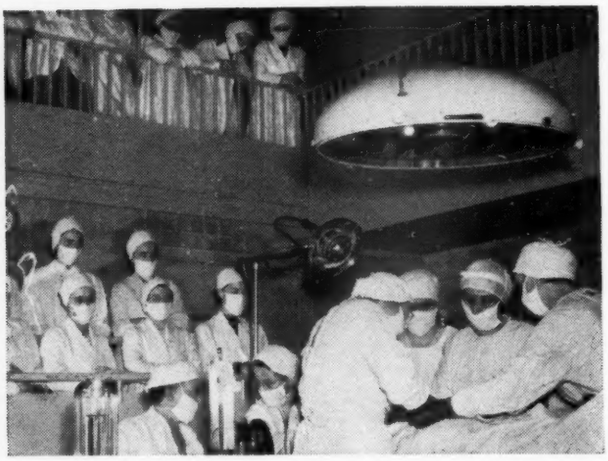
Roualle, H. L. M.: **Brit. M. J.** 2:1293 (Dec. 13) 1952.

Tucker, C. C.: **J. Kansas M. Soc.** *53*:230, 1952.

Weinberg, T.: **J. Sinai Hosp., Baltimore** *1*:21, 1952.


FOUGERA E. FOUGERA & COMPANY, INC · 75 Varick St., New York 13, N. Y.

*T. M.



*"Preoperatively* SULFASUXIDINE *is the drug of choice in preparing the colon."*[1]

For safer enteric surgery,

and speedier convalescence

# Sulfasuxidine®

SUCCINYLSULFATHIAZOLE

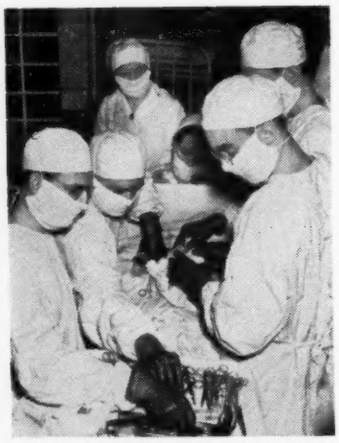
remains a drug of choice

for producing and maintaining

a sterile bowel.

0.5-GM. TABLETS, BOTTLES OF 100, 500 AND 1,000.

POWDER, ¼-LB. AND 1-LB. BOTTLES.



SULFASUXIDINE *". . . not only lowers the bacterial count, but keeps the stools somewhat soft and liquid."*[2]

SULFASUXIDINE *also maintains a low enteric bacterial count postoperatively. Gas pains are diminished, nausea and vomiting seldom occur, and recovery is quicker and less complicated.*

Sharp & Dohme

*Philadelphia 1, Pa.*

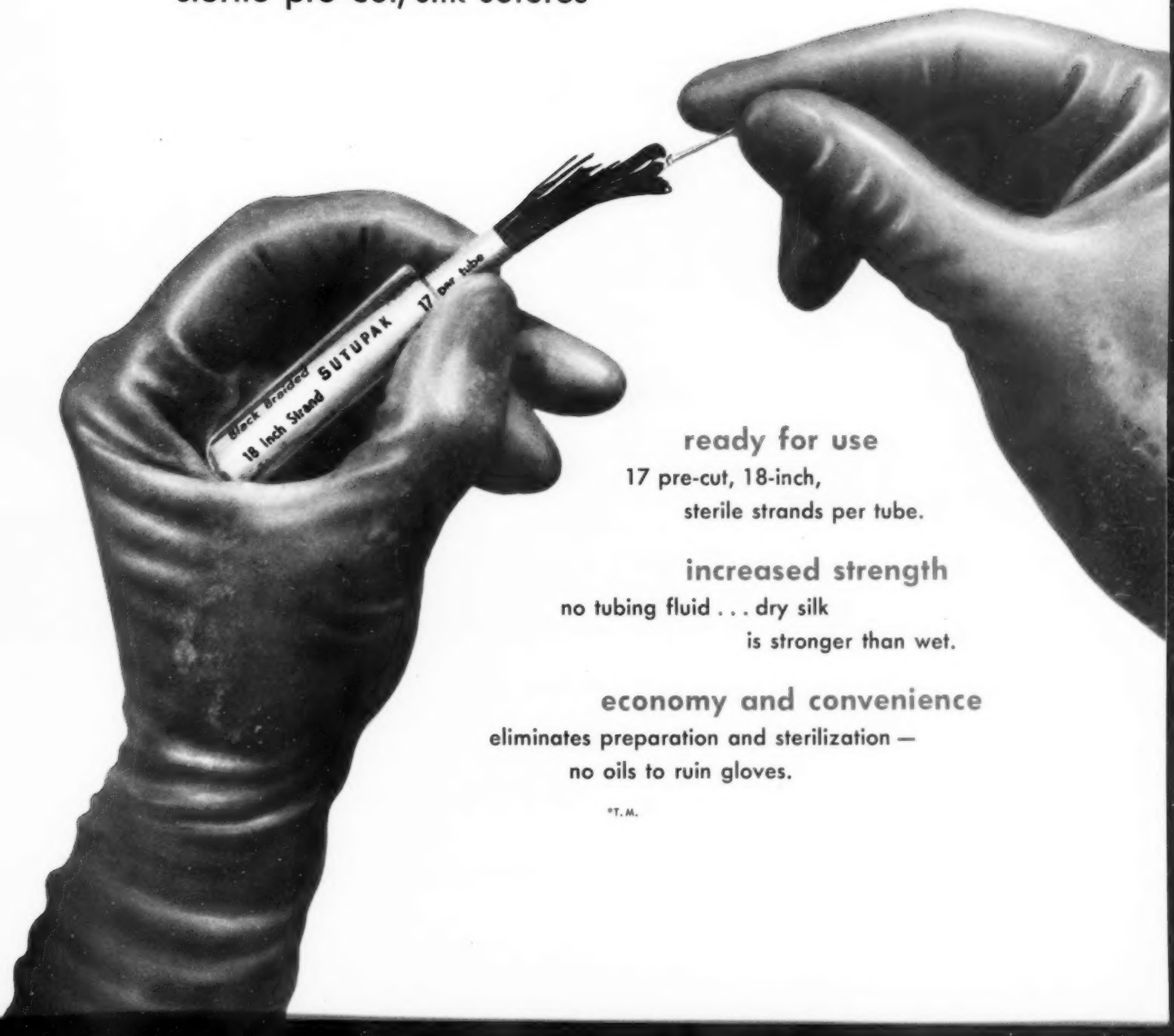
1. Weigel, C.J.: Am.J. Proctology, *1*:183, 1950
2. Halligan, E.J., Perkel, L.L. and Catlan, J.K.: Am. J. Proctology, 1950

*New*

# ETHICON®

# SUTUPAK*

sterile pre-cut, silk sutures




**ready for use**

17 pre-cut, 18-inch,
sterile strands per tube.

**increased strength**

no tubing fluid . . . dry silk
is stronger than wet.

**economy and convenience**

eliminates preparation and sterilization —
no oils to ruin gloves.

*T. M.

# *Uniform Absorption*

**ETHICON®** *Catgut*

Ethicon Tru-Chromicized catgut is absorbed at a remarkably uniform rate, regardless of suture size.

always specify **ETHICON** *Sutures*

**ETHICON SUTURE LABORATORIES INCORPORATED, NEW BRUNSWICK, N.J.**

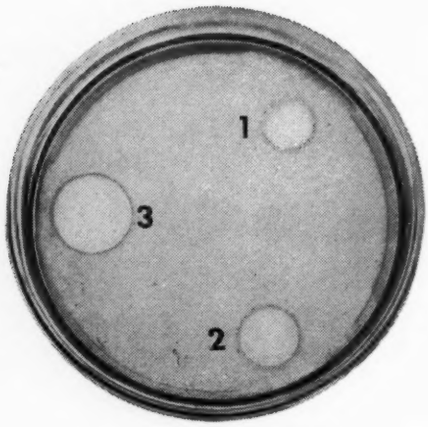
Upjohn

# preoperative bowel preparation within 24 hours:

# Neomycin Tablets

**Each tablet contains 0.5 Gm. neomycin sulfate (equivalent to 0.35 Gm. neomycin base)**
**Available in bottles of 20 tablets**

THE UPJOHN COMPANY, KALAMAZOO, MICHIGAN




Inhibitory action of penicillin (*1*), streptomycin (*2*), and PENSTREP (*3*), on a mixed culture of gram-positive and gram-negative organisms.

# For Wide-Spectrum Bactericidal Action

| PENSTREP "4:½" | PENSTREP "4:1" |
|---|---|

PENSTREP supplies a total of 400,000 units of Crystalline *Penicillin* together with Crystalline *Dihydrostreptomycin Sulfate*—the purest form of dihydrostreptomycin available. PENSTREP provides potent, synergistic bacteri*cidal* action that is effective over a *wide-range* spectrum.

PENSTREP "4:½" contains ½ Gm. of dihydrostreptomycin; PENSTREP "4:1" contains 1 Gm. It is recommended that "4:1" be used for short, intensive periods of therapy, "4:½" for prolonged treatment periods.

*Supplied:* PENSTREP "4:½": dry mixture for aqueous injection; also, aqueous suspension, ready to inject.

PENSTREP "4:1": dry mixture for aqueous injection.

All product forms available in one- and five-dose vials.

# PenStrep*

*PENSTREP is a trade-mark of Merck & Co., Inc.

*Research and Production for the Nation's Health*

MERCK & CO., INC.
*Manufacturing Chemists*
RAHWAY, NEW JERSEY
In Canada: MERCK & CO. Limited—Montreal

© Merck & Co., Inc.

# *Heaney* HYSTERECTOMY *Instruments*

*GS-205* HEANEY-SIMON Retractor. Improved model, with blade end slightly curved, comfortable handle. Blade 1″ x 4½″ on 10½″ shaft gives maximum exposure yet occupies minimum space. Stainless. Each, *$13.00*

*GS-210* HEANEY Retractor. Small size, blade 1″ x 1¾″. Stainless. Each, *$5.00*

*GS-211* Same, medium, 1⅛″ x 2⅞″. *$5.00*

*GS-212* Same, large, 1⅛″ x 3¼″. *$5.00*

*GS-810* HEANEY Hysterectomy Forceps. Standard model. Slender shafts and short, full curved, tapering jaws. Serrations cut on bias, with additional wide tongue and groove for secure hold. 7½″. Box lock. Stainless. Each, *$16.00*

Order Direct from

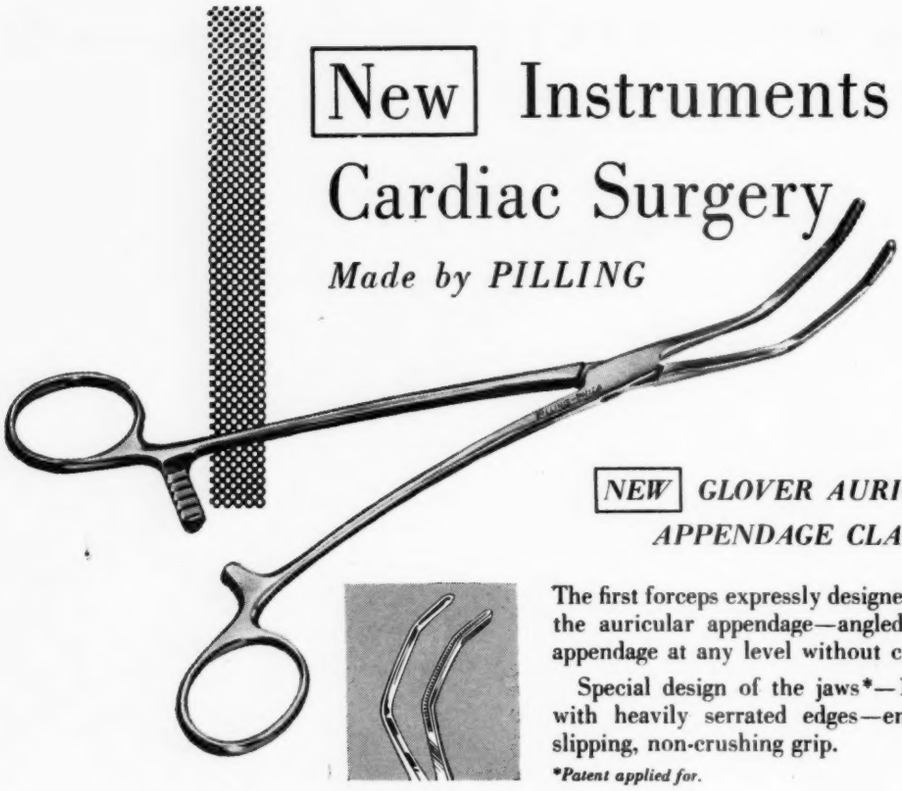
## *V. Mueller & Co.*

***Instrument Makers To The Profession Since 1895***

**330 SOUTH HONORE STREET** **CHICAGO 12, ILLINOIS**

# New Instruments for Cardiac Surgery

*Made by PILLING*



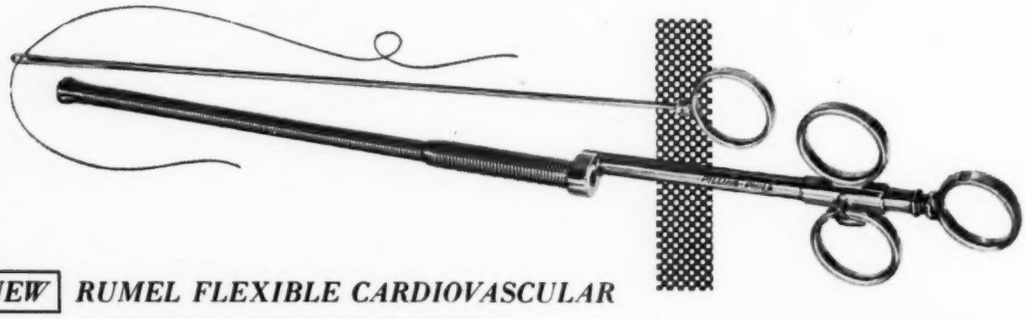
**NEW** ***GLOVER AURICULAR APPENDAGE CLAMP***



The first forceps expressly designed for clamping the auricular appendage—angled to grasp the appendage at any level without crushing.

Special design of the jaws*—1 x 2 grooved with heavily serrated edges—ensures a non-slipping, non-crushing grip.

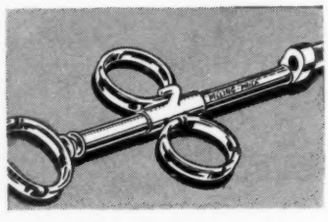
**Patent applied for.*



**NEW** ***RUMEL FLEXIBLE CARDIOVASCULAR TOURNIQUET with "eyed threader" for pulling suture through cannula.***

Specially designed to control tension on the auricular purse-string suture during mitral commissurotomy.

3-ring control and flexible cannula allow proper tension to be maintained with assistant's hands completely out of the wound.



*Write for prices and descriptive literature which gives complete details.*

Order direct from:

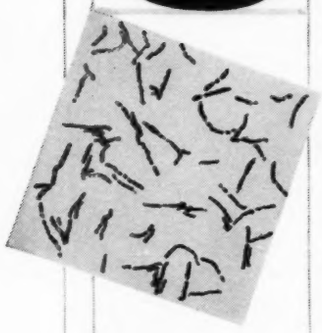
**GEORGE P. Pilling & SON CO.**

**3451 WALNUT STREET • PHILADELPHIA**

● **Headquarters for instruments for cardiac surgery**

# to be sure

# S-R® in penicillin therapy...

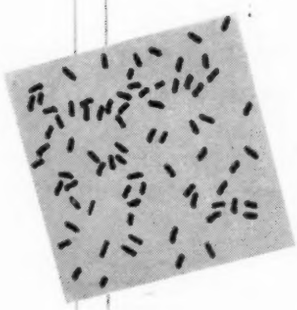


Parke-Davis procaine penicillin and buffered crystalline penicillin for aqueous injection

The high initial and prolonged effective levels achieved with S-R help control infections more certainly and minimize occurrence of drug-fast strains.

# S-R-D® in mixed or resistant infections...

Parke-Davis penicillin and dihydrostreptomycin



Combined antibiotic therapy with S-R-D gives *broad spectrum coverage* against many gram-positive and many gram-negative organisms—plus *cross fire action* against organisms sensitive to S-R and to dihydrostreptomycin.

## packaging

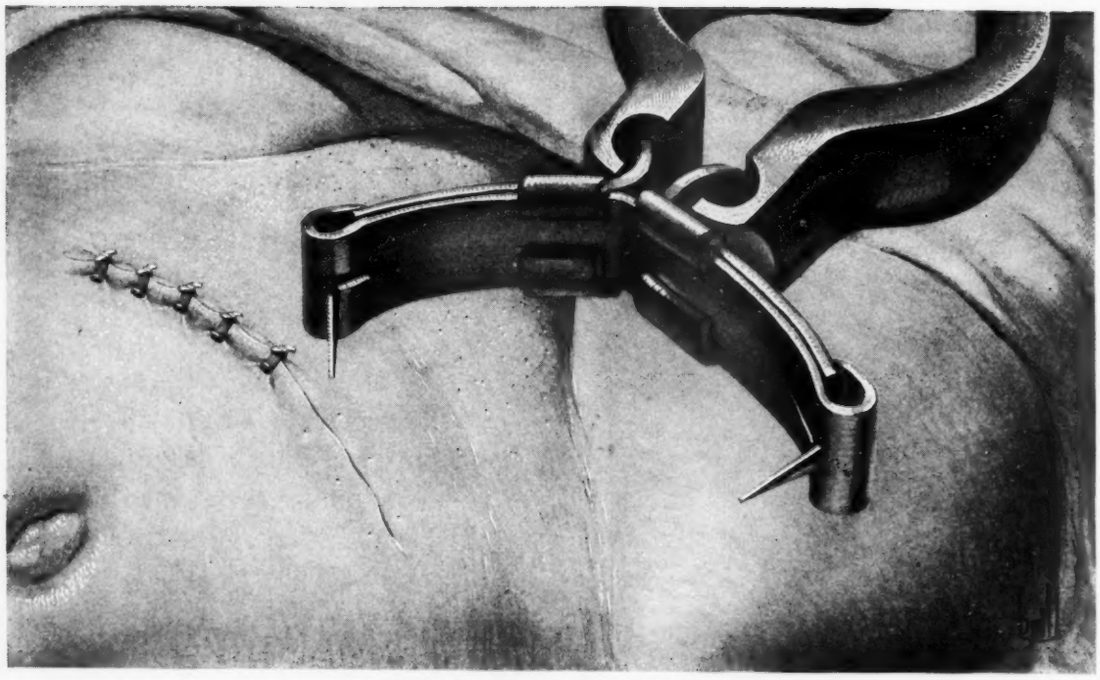
S-R is supplied in one-dose, five-dose and ten-dose Steri-Vials. Prepared as directed, each 1 cc. contains 400,000 units of S-R (300,000 units of procaine penicillin-G and 100,000 units of buffered crystalline penicillin-G).

S-R 1,000,000 units (one-dose Steri-Vial®) and S-R 10,000,000 units (ten-dose Steri-Vial) when prepared as directed, contain 1,000,000 units of S-R in each 1.25 cc.

S-R-D ½ Gm., and S-R-D 1 Gm. are supplied in one-dose and five-dose Steri-Vials. Prepared as directed, each dose provides 400,000 units of S-R and either ½ Gm. or 1 Gm. dihydrostreptomycin.

MEDICAMENTA VERA

Parke, Davis & Company
DETROIT, MICHIGAN



Removal of Serature "Spur" wound clip.

# "They are so easy to remove... patients really appreciate them."

## ...SO EASY TO APPLY—AND PRODUCE SUCH EXCELLENT RESULTS— SURGEONS APPRECIATE THEM TOO

Only Propper's Serature Wound Clips have the unique, integral "spurs" for precise adjustment after primary application and quick, painless removal. Only Serature Wound Clips give you all these features:

- *needle sharp teeth, perfectly aligned, for speedy application, accurate closure, and minimal tissue trauma*
- *faster healing, reduced incidence of "stitch" infection and better cosmetic results*
- *speedy and pain-free "single-step" removal, at the identical angle as that of application*
- *economy of use because they're removed intact and are "pore sealed" to render them chemically inert and impervious to body fluids and sterilizing procedures . . . usable over and over again*
- *applied and removed with ordinary wound clip forceps—no special instruments required*

Application of Serature clip.

**PROPPER MANUFACTURING CO., INC.**
**10-34 44th Drive, Long Island City 1, N. Y.**
*Specialized Quality Products for the Laboratory, Hospital and Physician*

Have your O.R. Supervisor specify

# Propper Serature "Spur" Wound Clips

***The Modern Closure Technique***

READY-MADE

# Vitallium

## HIP PROSTHESES

**SIX DESIGNS • CLINICALLY PROVEN**
**STRONG • INERT • LONG WEARING**

All six designs of Vitallium hip prostheses are the result of careful thought, trial and study on the part of the initiating surgeon. All have been implanted successfully and their use is recommended in properly selected cases.

Vitallium hips offer a variety in design which permits selection of the type best suited to the patient's requirements. They are strong, well-designed and wear-resistant. The head portions are smoothly polished and except for the Lippmann type are hollow for lightness.

Like all Vitallium appliances, the suitability of these prostheses for permanent implantation has been proven throughout 15 years of use.

**VITALLIUM HIP DESIGNS**
*(from left to right)*

1. Modified Judet Type No. 6902
2. F. R. Thompson Type No. 6936
3. Moore Type No. 6939
4. J. E. M. Thompson Type No. 6938
5. McBride Type No. 6935
6. Lippmann Type No. 6932

* * *

WRITE FOR MORE DETAILED INFORMATION, INCLUDING SIZE DATA AND SUGGESTED OPERATING TECHNIQUES.

***ORDER THROUGH YOUR SURGICAL DEALER***

AUSTENAL **SURGICAL DIVISION** LABORATORIES, INC.

224 EAST 39th STREET • NEW YORK 16, N. Y.

## *To Hasten Recovery*

**'Pancebrin' is the choice multivitamin combination for the debilitated or surgical patient. Each 2 cc. contain:**

| | |
|---|---|
| Thiamin Chloride | 10 mg. |
| Riboflavin | 2 mg. |
| Pyridoxine Hydrochloride | 3 mg. |
| Pantothenic Acid (as Sodium Pantothenate) | 3 mg. |
| Nicotinamide | 20 mg. |
| Ascorbic Acid | 60 mg. |
| Alphatocopherol | 2 mg. |
| Vitamin A | 10,000 U.S.P. units |
| Vitamin D | 1,000 U.S.P. units |

**'Pancebrin' may be injected intramuscularly without dilution; or, if desired, it may be added to dextrose and physiological saline intravenous infusions. The dose is usually 2 cc. daily, continued as long as needed.**

*Eli Lilly and Company*
*Indianapolis 6, Indiana, U. S. A.*

Lilly

***Specify the 2-cc. ampoules or the 10-cc. rubber-stoppered ampoules—available at pharmacies everywhere.***

AMPOULES

# 'Pancebrin'

(MULTIPLE VITAMINS, INJECTABLE, LILLY)

The American Journal of Surgery

Copyright, 1953 by The American Journal of Surgery

A PRACTICAL JOURNAL BUILT ON MERIT

Sixty-second Year of Publication

VOL. LXXXV MARCH, 1953 NUMBER THREE

# Presidential Address

## THE FUTURE OF THE TREATMENT OF TRAUMA

ARTHUR R. METZ, M.D.

*Chicago, Illinois*

THIS is the fifteenth meeting of the American Association for the Surgery of Trauma and time to evaluate the progress that has been made since the organization meeting on June 14, 1938, and to plan for the future.

At the Founders' meeting Dr. Edgar L. Gilcreest, Chairman of the committee of organization, outlined in detail the object for such a society to be formed, to study and advance the whole science of the Surgery of Trauma. He appealed to the general surgeon and to the allied specialties to cooperate in this important and increasing field of surgery. There were sixty-eight surgeons present who were designated the Founder Fellows. The original quota of 200 was soon filled and with the increased interest in the association, the quota was increased to 250. An analysis of the Founder Fellowship group after fourteen years shows that twenty-three are still on the Active roll, twenty-three have been transferred to Senior Fellows and twenty-two have died.

The first presidential address was delivered by Dr. Kellogg Speed at Hot Springs, Virginia, on May 8, 1939, and was a learned discussion of the rapidly expanding field of general surgery, and the breaking-off of certain specialties into narrow fields, with the formation of American Boards.

The Directory of Medical Specialists lists nineteen Boards of which ten represent some field of surgery as: neurologic surgery, obstetrics and gynecology, ophthalmology, orthopedic surgery, otolaryngology, plastic surgery, proctology, surgery, thoracic surgery and urology.

Dr. Speed designated the Association as a high-standing forum for discussion of the problems of trauma in surgery, and an opportunity to sift the merits of advances devised or reported.

The addresses of the former Presidents have contributed much for the cultivation and improvement of the science and art of the surgery of trauma and allied sciences, and the programs have covered every phase of the field of trauma. Time will continue to help achieve the objectives and standards planned by the Founders.

Plans for the future of the treatment of trauma must continue as was well expressed by Dr. Casper Hegner in his presidential address in 1948, "We must be alert and become belligerently aggressive if we are to justify the function and purpose of our organization. Our duty is clear, the task is great and the program formidable. With unity of purpose, we can accomplish great things for surgery and the better service for mankind."

We should work aggressively toward a train-

ing program that begins at the student level and continues through the internship and residency period. To accomplish this the responsibility rests with the surgical departments and deans of all medical schools.

There should be established in the surgical department a sub-department designated as the surgery of trauma under a chairman and staff, who will coordinate with the other surgical specialties the many problems that are found in the injured patient so that prompt and necessary treatment can be given and rehabilitation started early during the course of recovery.

The problems of surgical teaching in hospitals was thoroughly discussed last year by Dr. Arnold Griswold in his presidential address, and need not be repeated. Much good clinical material is overlooked for teaching purpose in the outpatient departments of many hospitals and dispensaries where industrial patients report for examination and treatment. The importance of a good history, complete examination and careful record must be emphasized. What appears to be a minor injury may become a major problem which fact should repeatedly be impressed upon the young doctor.

The analysis of accidents published by the National Safety Council in Accident Facts—1952 gives some very useful and impressive information as to the number of accidents, deaths and costs to the nation in 1951. To quote briefly some of these facts will better impress the surgical profession, medical schools and hospitals of the importance of the surgery of trauma.

In 1951, there were reported 94,000 accidental deaths and 9,400,000 non-fatal injuries. total occupational deaths while at work, 16,000 and 2,100,000 injured; total motor-vehicle deaths in motor-vehicle accidents, 37,300 and 1,300,000 injured; total home deaths 28,000 and 4,200,000 injured in home accidents; total public non-occupational non-motor-vehicle deaths, 15,000, and 1,900,000 injured—an increase of 7 per cent.

Costs of accidents in 1951 in which deaths and injuries occurred, together with non-injury motor-vehicle and occupational accidents and fires, cost the nation at least $7,900,000,000.

These costs include:

| | |
|---|---|
| Minimum estimates of wages lost by disabled persons, lower wages when returned to work due to permanent partial disability, present value of future earnings of those permanently incapacitated or killed in accidents | $2,900,000,000 |
| Medical fees, hospital expense | 550,000,000 |
| Administrative and claim settlement costs of insurance | 1,000,000,000 |
| Property damage in motor-vehicle accidents | 1,400,000,000 |
| Property destroyed by fire | 731,000,000 |
| Property destroyed or production lost due to occupational accidents | 1,300,000,000 |

Leading causes of death in age groups in 1949 showing the death rate per 100,000 population as follows:

| Age in Years | | Death Rate per 100,000 Population |
|---|---|---|
| 1–5 | Accidents | 39 |
| | Pneumonia | 18 |
| | Congenital malformations | 12 |
| 5–15 | Accidents | 23 |
| | Cancer | 6 |
| | Poliomyelitis | 4 |
| 15–25 | Accidents | 52 |
| | Tuberculosis | 15 |
| | Cancer | 9 |
| 25–45 | Heart disease | 51 |
| | Accidents | 44 |
| | Cancer | 40 |
| 45–65 | Heart disease | 527 |
| | Cancer | 271 |
| | Vascular lesions | 125 |
| | Accidents | 61 |
| 65 and over | Heart disease | 2,771 |
| | Vascular lesions | 883 |
| | Cancer | 842 |
| | General arteriosclerosis | 235 |
| | Accidents | 222 |

This Association with fourteen years of experience to its credit, specializing in the Surgery of Trauma, can now consider what its program should be for the future. The trend of modern times is to designate minimum qualifications and standards for a surgeon to have in order to treat different types of surgical conditions as is provided by the ten Boards already organized.

The logical answer is to organize a Board for the Surgery of Trauma—with this Association as the Founder Group—along with the cooperation of the American Board of Surgery and

the other Boards dealing with surgical problems, joining in the teaching and training of students and young doctors.

Accident Facts shows that injuries increased 4 per cent in 1951 as compared to 1950, and cost the nation over $7,900,000,000 in 1951, which is evidence that trauma is one of the surgeons' major problems which requires special supervision to improve the science and art of the Surgery of Trauma.

The National Safety Council which is a non-profit cooperative organization representing many of the country's largest industries invites such a program and will lend their support.

The special committee on Investigation of Practices in Hospitals regarding restrictions on surgery, and results of questionnaire on the care of trauma which was reported by Committee Chairman, Dr. George Curry, at the executive session in 1951, contains much valuable information with recommendations which have been approved by the American Association for the Surgery of Trauma.

A committee to study the problems attendant upon organizing an American Board for the Surgery of Trauma has been active since our last meeting and will submit its report at the Executive Session.

As an officer of this Association since its organization it has been a great satisfaction to see it develop into one of the leading surgical groups of the nation, and I now urge that we continue aggressively to plan to achieve to a greater degree the precepts as stated in the Constitution. We should seek to develop in the Department of Surgery of all Class A Medical Colleges, a sub-department for the teaching of the Surgery of Trauma.

A Board for the Surgery of Trauma should be organized as an affiliate of the American Board of Surgery.

I have been honored to serve the Association as Secretary at the organization meeting, Treasurer for ten years, Chairman of the Constitution and By-Laws Committee, and President. In working together for the good of a common cause one makes many lasting friendships.

I will end my official duty at the close of this meeting and fade away like an old surgeon, soldier and sailor who has dedicated his professional life to the "relief of suffering and prolongation of life."

# Scientific Papers

## FRACTURE OF THE NECK OF THE HUMERUS WITH DISLOCATION OF THE HEAD FRAGMENT*

CHARLES S. NEER, M.D., THOMAS H. BROWN, JR., M.D.
AND HARRISON L. MCLAUGHLIN, M.D.
*New York, New York*

THIS paper presents a series of fracture-dislocations of the shoulder treated by the Fracture Service of the New York Orthopaedic—Columbia-Presbyterian Medical Center between the years 1929 and 1952. The literature upon this subject is rather small and it is disappointing to find that authors vary in opinion as to what a "fracture-dislocation" of this joint really is. This, of course, leads to confusion regarding results of treatment. For example, one article[1] has been widely quoted in support of closed manipulative reduction. The author is said to have been successful with this method in six of seven fracture-dislocations. When one actually studies these cases, it is apparent that three of them were straight forward "surgical neck" fractures and three other cases, in which dislocation was shown to be present, the fracture was impacted with little displacement. It is, therefore, appropriate to correlate terminology and pathology before going on to the details of treatment and results.

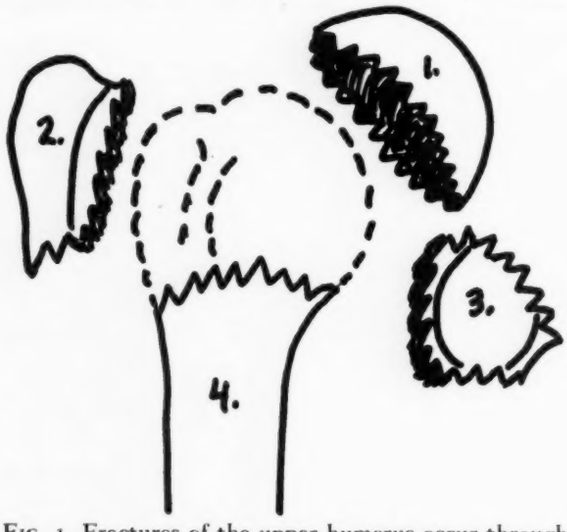



FIG. 1. Fractures of the upper humerus occur through lines of epiphyseal union. Four fragments result: (1) head, (2) greater tuberosity, (3) lesser tuberosity, (4) shaft.

### TERMINOLOGY

Codman[2] suggested that fractures of the neck of the humerus occurred through approximately the lines of epiphyseal union. (Fig. 1.) Four major fragments result: (1) The anatomic head, (2) the greater tuberosity, (3) the lesser tuberosity and (4) the shaft. Figures 3 and 4 illustrate the validity of his conclusion. It is, therefore, confusing to attempt, as some writers have done, to distinguish "surgical neck fracture-dislocations" from "anatomic neck fracture-dislocations." The term "humeral neck fracture" is perhaps more accurately descriptive. The four-fragment pattern varies only in the degree of displacement of the individual fragments.

It seems proper to emphasize a second point made by Codman.[2] The mechanism of injury, namely, locking of the humerus against the acromion in the "pivotal position," is precisely the same for both fractures of the humeral neck and subcoracoid dislocations of the gleno-humeral joint. Younger subjects usually dislocate rather than fracture, whereas, in older individuals the bone generally gives way. The latter is presumably due to the relative weakness of bone in the aged. It is probable that many of these fractures are associated with significant soft part damage and are in a sense fracture-dislocations spontaneously reduced. (Group 1, Fig. 2.) It is not possible to estimate

* From the Department of Orthopaedic Surgery, College of Physicians and Surgeons, Columbia University, and the Fracture Service, Presbyterian-New York Orthopaedic Hospitals, New York, N. Y.

the number of lesions of this sort but it must be quite large. (Table 1.)

As the title suggests, this paper concerns itself only with twenty patients in whom the head fragment remained extruded for roentgen proof of dislocation. Group 1 (Fig. 2) illustrates

TABLE 1
INCIDENCE—FRACTURE-DISLOCATION, 1929–1951

| | No. | Per cent |
|---|---|---|
| Fr. neck humerus | 921 | 51.2 |
| Dislocation | 855 | 47.7 |
| Anterior 759 | | |
| Posterior 25 | | |
| Fr. tuberosity 71 | | |
| Fr. neck with dislocation | 20 | 1.1 |
| Anterior 17 | | |
| Posterior 3 | | |
| | 1,796 | 100 |

lesions which have been described as "fracture-subluxation,"[3] "abduction fracture-dislocation,"[4] "comminuted fracture-dislocation,"[5] "fracture with dislocation of the shaft"[6] and by various other terms. It is not intended to discuss Group 1 further, this has been dealt with elsewhere,[7–8] but rather to concentrate upon seventeen anterior and three posterior Group 11 lesions.

### ANTERIOR (SUBCORACOID) FRACTURE-DISLOCATIONS

*Pathology.* The fracture remained impacted in only two of seventeen subcoracoid displacements. In fifteen patients the head fragment had been extruded through the capsule and remained in the "upside down" position as the arm returned to the side. These lesions present a remarkably constant picture, as illustrated in Figures 3 and 4. The head is detached, extracapsular, and often rotated as much as 180 degrees. The greater tuberosity is retracted by the external rotators, the lesser tuberosity remains attached to the subscapularis and the shaft is pulled upward by the long muscles crossing the shoulder joint. The separation of the tuberosities uproots the long tendon of the biceps. The head fragment is essentially devoid of soft-part attachments and, so Largomarsino[9] has suggested, is even more susceptible to avascular necrosis than the femoral head in that it lacks a ligamentum teres. The head fragment rests upon the brachial plexus (Fig. 5), making surgical approach difficult unless executed from above downward.[7]

*Clinical.* The average age was, interestingly, midway between young and old, being fifty-six years. One might expect double lesions to occur in this age group. The history invariably was that of a very heavy fall and the patient was often obese. The left shoulder was involved in thirteen of seventeen injuries. In contrast to many textbook statements, nerve and vascular injury was unusual with transient brachial plexus symptoms occurring in only two patients and in no instance was operation forced by major-vessel injury.[10] (Table 11.)

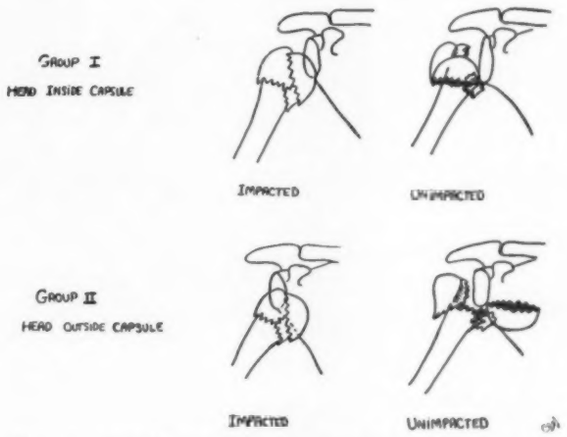



FIG. 2. Classification of humeral neck fracture-dislocations.

TABLE II
CLINICAL—SEVENTEEN ANTERIOR

| | |
|---|---|
| Ave. age | 56 yr. |
| Patient | (often obese) |
| Mechanism | Hard fall (details vague) |
| Down stairs | 4 |
| From train | 2 |
| On ice | 2 |
| Other "fall" | 7 |
| Hit by car | 2 |
| Neurovascular damage | (unusual) |
| Nerve | 2 |
| Major vessel | 0 |

### POSTERIOR (SUBSPINOUS) FRACTURE-DISLOCATION

*Pathology.* The anterior portion of the articular surface of the humerus is often gouged out by impingement against the posterior glenoid in subspinous dislocations.[11] However, the entire anatomic head was separated from the humerus and remained posterior in three of twenty-five posterior dislocations. (Fig. 6.) In

FIG. 3. A, anteroposterior view and B, lateral view of a typical unimpacted anterior fracture-dislocation; note the four fragments.

FIG. 4. Two other unimpacted anterior fracture-dislocations. Note constant pattern and resemblance to Figure 3.

two of these patients the lesser tuberosity had, in addition, been avulsed by the taut subscapularis. The head fragment retained few if any soft-part attachments. Figure 7 illustrates the value of obtaining lateral or axillary roentgen views as well as the usual anterior-

TABLE III
CLINICAL—THREE POSTERIOR

| | |
|---|---|
| Ave. age | 36 yr. |
| Mechanism | Arm below shoulder level |
| Electrician (shock) | 1 |
| Epileptic (attack) | 1 |
| "Fell on ice" | 1 |
| Neurovascular damage | none |

posterior when evaluating posterior glenohumeral displacements.

*Clinical.* The mechanism of these injuries is made obscure by the fact that two of the patients were rendered unconscious. One can assume internal rotation leverage with the arm below shoulder level. (Table III.) All three patients were muscular young males.

TREATMENT

Table IV lists the possible methods of treatment and indicates the experience with each.

TABLE IV
TREATMENT—TWENTY FRACTURE-DISLOCATIONS

| | |
|---|---|
| 1. Do nothing | 0 |
| 2. Closed reduction | 6 |
| 3. Open reduction | 3 |
| 4. Removal head | 16 |
| Simple removal—8 | |
| With reconstruction—8 | |
| 5. Fusion | 1 |
| 6. Replacement prosthesis | 0 |

Closed reduction was performed upon six occasions and was successful in only the two instances in which the fracture was impacted. The head fragment was subsequently removed in the four failures. Open reduction was ac-

complished in three unimpacted fractures, the head and tuberosities being attached to the shaft by means of suture wire on the bicepital tendon. In one patient, the head fragment slipped out of position two days after open reduction and was removed two weeks later.

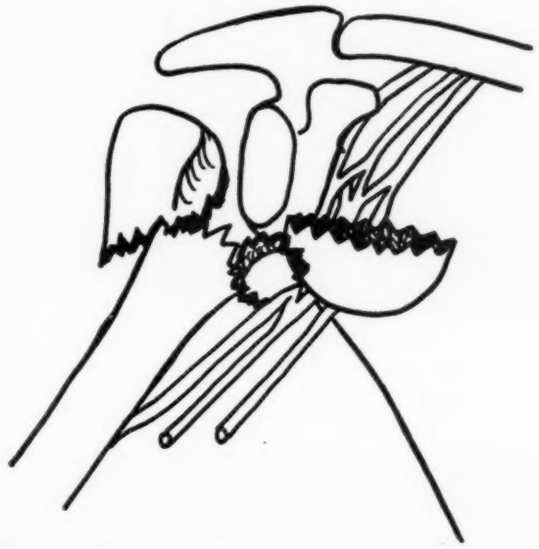


FIG. 5. Unimpacted anterior fracture-dislocation. Note the head fragment extracapsular resting upon the brachial plexus. The retraction of the tuberosities exposes biceps tendon.

A second required removal of the head for control of infection. Primary arthrodesis was attempted in one of the earlier cases, however, this procedure did not invite repetition. Ostectomy for removal of the humeral head was performed upon sixteen patients and in eight of these an additional reconstructive procedure was executed, e.g., transposition of the cuff, biceps tendon suspension, excision of acromion, or excision of outer end of clavicle. Although replacement prosthesis is currently being investigated, this method was not employed in this series.

## RESULTS

Physical evaluation and radiologic examination was maintained for an average period of six years after treatment. Table v indicates the length of follow-up.

One of the impacted subcoracoid lesions treated by *closed reduction* obtained an almost perfect result while the result of the second impacted fracture-dislocation was compromised by the rapid development of avascular necrosis of the head fragment. Closed reduction proved inadequate and injudicious in four unimpacted lesions. *Open reduction* resulted in failure consistantly in three unimpacted lesions. Figure 8 illustrates the preoperative, six-week postoperative and sixteen-year follow-up result of an open reduction indicating severe avascular

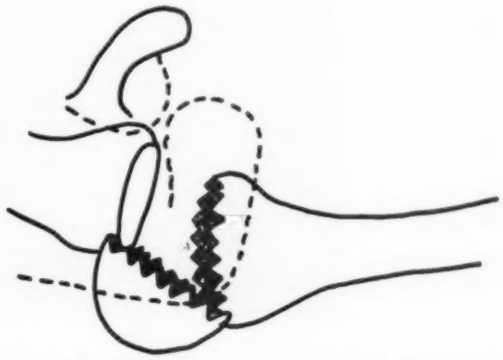


FIG. 6. Axillary view of an unimpacted posterior fracture-dislocation. Head fragment remains posterior and lesser tuberosity is often avulsed (not shown).

necrosis. Two other open reductions developed early complication, already described, requiring excision of the head.

*Resection of the head* removed the fulcrum necessary for glenohumeral abduction and rotation. During the early months following this procedure the shoulder was flail, but with progressive fibrosis and ossification glenohumeral motion became restricted. Figure 9 demonstrates the preoperative and six years' result of simple excision of the head. Note the acromial impingement upon full abduction due to the absence of the fulcrum. External rotation was usually less than zero due to posterior displacement of the greater tuberosity. Fatigue pain was the major complaint. Five of the nineteen resections developed solid bony bridging from humerus to scapula. Table VI describes various reconstructive procedures performed with head removal and, contrary to previous

TABLE V

FOLLOW-UP—TWENTY FRACTURE-DISLOCATIONS

| | |
|---|---|
| 4 mo.–2 yr | 7 |
| 2–4 yr | 1 |
| 4–6 yr | 1 |
| 6–8 yr | 4 |
| 8–10 yr | 3 |
| 10–12 yr | 1 |
| 12–14 yr | 2 |
| 14–16 yr | 0 |
| 16–18 yr | 1 |

Average follow-up—6 yrs.

7A 7B

FIG. 7. A, anteroposterior view and B, lateral view of unimpacted posterior fracture-dislocation. Axillary and lateral roentgenograms are essential to the evaluation of posterior displacements.

8A 8B 8C

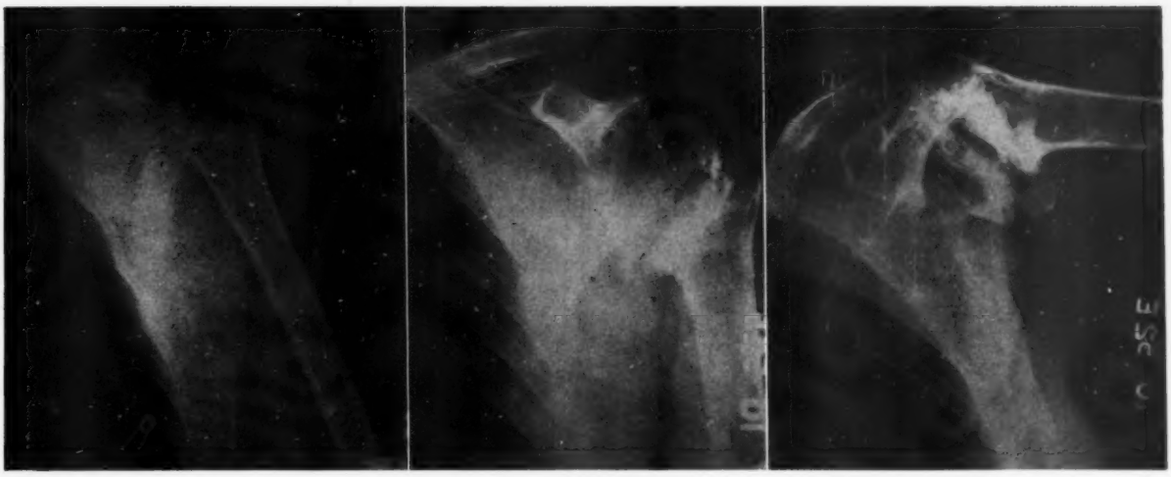
FIG. 8. A, preoperative; B, six-week postoperative and C, sixteen-year follow-up result of an open reduction. Note late necrosis of the head.

impression,[7,8–12] it was concluded that plastic procedures upon the musculotendinous cuff or the acromion did not improve the result. There was usually from 5 to 25 degrees glenohumeral motion following head removal regardless of whether or not a plastic procedure had accompanied the resection. In one patient excision of the outer end of the clavicle seemed to be a worth while adjunct to head removal. Time lapse from injury to operation[13] (seven patients within twenty-four hours, four patients within ten days, seven patients from ten days to six months) did not definitely effect the result nor did postoperative management. The muscle power and personality of the patient seemed to be the major determining factors. In spite of these handicaps, fourteen of nineteen resection patients were satisfied with their result and were carrying out their usual work without appreciable disability.

TABLE VI

REMOVAL HUMERAL HEAD—NINETEEN CASES

| | |
|---|---|
| Simple removal | 8 |
| Excision acromion | 5 |
| Transpose cuff (tuberosities) | 5 |
| Biceps tendon suspension | 2 |
| Excision outer end clavicle | 1 |



9A 9B 9C

FIG. 9. Simple excision of the head fragment. A, preoperative; B and C, six-year result showing maximum abduction. Note impingement of the humerus upon the acromion due to the absence of the fulcrum of the normal head.

*Primary arthrodesis* was attempted in only one patient proving to be a difficult procedure, requiring long postoperative immobilization and resulting in fusion failure.[14]

Table VII sums up the results. Simple removal of the head seemed to be the best procedure to date for unimpacted lesions. Nevertheless, the limited motion and fatigue pain following resection has suggested the possible value of a *replacement prosthesis* to serve as a fulcrum for motion. Figure 10 depicts an attempt to achieve such an instrument. This was only recently devised and has not as yet had adequate trial.

TABLE VII
SUMMARY RESULTS—TWENTY FRACTURE-DISLOCATIONS

| | Failure | Work |
|---|---|---|
| 6 Closed reductions | 5 | .... |
| 3 Open reductions | 3 | .... |
| 16 Removal head | 0 | 5 mo. |
| 1 Fusion | 1 | 1 yr. |

FIG. 10. A recently devised articular replacement currently being investigated.

## CONCLUSIONS

1. Twenty fractures of the neck of the humerus with dislocation of the head fragment are presented. The pathology and terminology are correlated.

2. Primary arthrodesis is rendered difficult by humeral neck fracture.

3. If the fracture is impacted, the dislocation should be reduced by carefully performed open or closed methods, but with the knowledge that the head may subsequently undergo necrotic change.

4. The best treatment to date for unimpacted lesions is simple excision of the head fragment.

5. Replacement prothesis presents logical possibilities and may prove of value in dealing with major injuries of humeral head. Its true worth remains to be determined.

REFERENCES

1. Bohler, L. Die Behandlung von Verrenkungobrischen der Schulter. *Deutsche Ztschr. f. Chir.*, 219: 238, 1929.
2. Codman, E. A. The Shoulder. Thomas Todd Co. Boston, 1934.
3. Fairbanks, T. J. Fracture-subluxations of the shoulder. *J. Bone & Joint Surg.*, 30: 454. 1948.
4. Watson-Jones, R. Fractures and Other Bone and Joint Injuries. Edinburgh, 1944. E. & S. Livingstone.
5. Michaelis, L. S. Comminuted fracture-dislocation of the shoulder. *J. Bone & Joint Surg.*, 26: 363. 1944.
6. Garré, C., Kuttner, H. and Lexer, E. Handbuch der Praktischen Chirurgie. Stuttgart, 1922. F. Enke.
7. McLaughlin, H. L. Treatment of Shoulder Injuries. Regional Orthopedic Surgery and Fundamental Orthopedic Problems. Ann Arbor, Mich., 1947. Edwards.
8. DePalma, A. E. Surgery of the Shoulder. Philadelphia, 1950. J. B. Lippincott.
9. Largomarsino, E. H. and Muscolo, D. T. Necrosis asepticia de la cabega humeral consecutiva a luxacion fracture de la misma. *Arch. urug. de med., cir. y. especialid.*, 17: 450. 1940.
10. Steindler, A. The Traumatic Deformities and Disabilities of the Upper Extremity. Springfield, Ill., 1946. Charles C Thomas.
11. McLaughlin, H. L. Posterior dislocation of the shoulder. *J. Bone & Joint Surg.*, 34: 584. 1952.
12. Jones, L. The shoulder joint. Observations on the anatomy and physiology with an analysis of a reconstructive operation following extensive injury. *Surg., Gynec. & Obst.*, 75: 433. 1942.
13. Guillermo, M. Fracture du col anatomique et transtuberositoire de l'humerus gauche avec luxation posterior-inferieuri irreductible. Resection precoci. *Soc. de méd. mil. franç., Bull. mens.*, 33: 272, 1939.
11. Thomsen, W. Zur Technick der operative Versteifung der Schulter nach Humeruskopfresektion. *Chirurg.*, 20: 531, 1949.

# PARTIAL CLAVICULECTOMY*

CARRUTH J. WAGNER, M.D.
*Philadelphia, Pennsylvania*

DURING the past eight years we have had occasion, for the treatment of a variety of lesions, to resect the outer third of the clavicle in forty-six instances. Sufficient time has elapsed to evaluate adequately this procedure and prompts the present report. A more than casual visit with the literature reveals this procedure to be an old one, well tried and evaluated.[1–9] Furthermore, recent studies by Inman[10,11] would suggest a wider indication for resection of the outer third of the clavicle than is commonly appreciated.

The average textbook ascribes the clavicle with the function of a flexible outrigger, serving as a prop for the shoulder and a strut for muscle attachments. Little or no mention is made of the intimate relationship between the clavicle, scapula and humerus and their associated joints. Codman observed the harmony of movement that existed in these joints and aptly named it "scapulo-humeral rhythm." The complicated clavicular motion in this "rhythm," however, was unsuspected until Inman and Saunders reported their magnificent experimental work.[11] To evaluate a procedure entailing partial removal of the clavicle one must, therefore, consider the shoulder as a unit and understand the inter-relationship of its various components in each phase of motion. Similarly any break in the rhythm of motion is positive evidence of derangement of one or more of the several components of the shoulder mechanism.

Inman has demonstrated that elevation of the arm, in either abduction or flexion, is accomplished by simultaneous motion in both the glenohumeral and scapulothoracic joints. The relationship during the first 30 degrees of elevation by abduction and 60 degrees by flexion, the so-called "setting phase," varies with the individual; but once these positions are reached, the relationship remains constant at a 2:1 ratio for every 15 degrees of motion, 10 are glenohumeral and 5 are scapulothoracic.

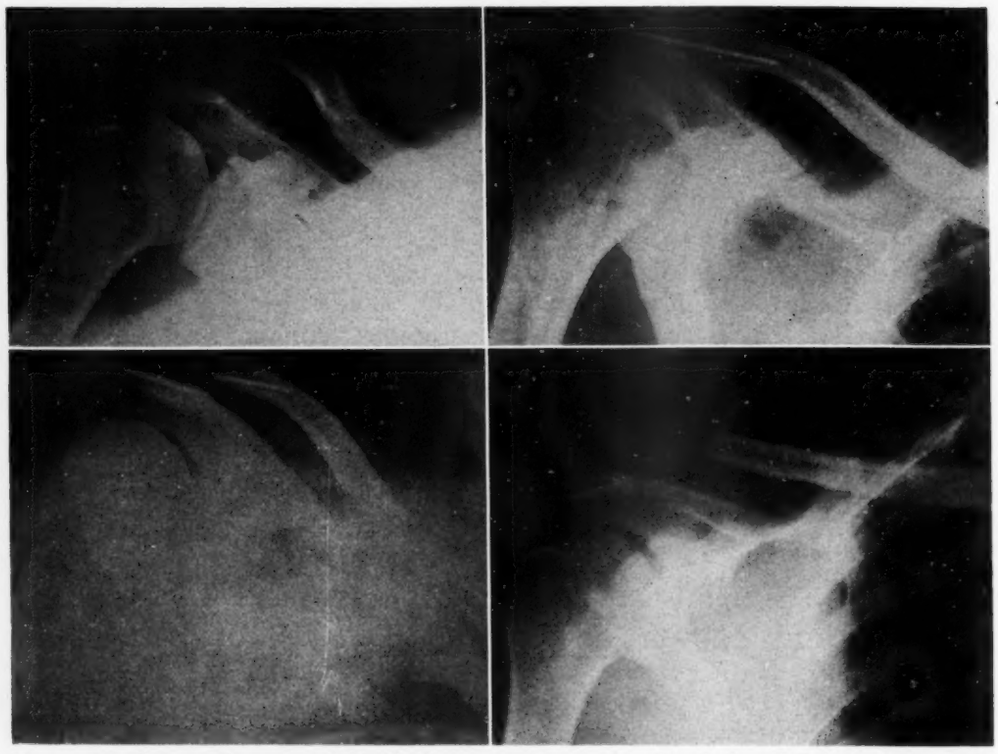
This continuous rotation of the scapula upon the thoracic cage is made possible by the total but unequal motion of the two clavicular joints. Inman[10] demonstrates that elevation of the arm is accompanied by elevation of the clavicle through the sternoclavicular joint. that this occurs early and is complete during the first 90 degrees of elevation, and that for every 10 degrees of elevation of the arm there are four degrees of elevation of the clavicle. When 90 degrees elevation is reached, motion practically ceases in the sternoclavicular joint.

TABLE 1
TOTAL CASES RESECTED

| | |
|---|---|
| Acromioclavicular separation | |
| Incomplete | 8 |
| Complete | 14 |
| Arthritis acromioclavicular joint | 12 |
| Tuberculosis, acromioclavicular joint | 2 |
| Non-union, fracture, clavicle | 2 |
| Fracture, distal end of clavicle | 3 |
| Arthrodesis, shoulder | 3 |
| Total | 46 |

Motion through the acromioclavicular joint on the other hand occurs early in the first 30 degrees, then ceases only to resume after 135 degrees of elevation are reached. To envisage this motion at the acromioclavicular joint one must keep in mind the two curves of the clavicle which allows it to rotate 50 degrees like a crankshaft. This rotation is consistent with intact coracoclavicular ligaments because they are attached to the apex of the lateral curve. Thus the clavicle, although rigidly attached to the scapula in its outer third, finds the total motion of its two joints equals the total motion of the scapula. It follows, therefore, that the scapula (and secondarily, the arm) will be limited in its total elevation by any limitation of motion of the acromioclavicular or sternoclavicular joints. The total elevation of the arm to 180 degrees is realized by 120 degrees motion in the glenohumeral and 60 degrees of scapular motion. The latter is realized by 30 degrees of elevation of the clavicle at the sternoclavicular joint and the

* From the Orthopedic Service, U. S. Public Health Service Hospital, Staten Island, N. Y.



1A 1B 1C 1D

FIG. 1. A to D, complete acromioclavicular separations, reduced and pinned. Injury three weeks postoperatively with fracture of the pin and redislocation; outer third of clavicle resected. In this case insufficient clavicle has been removed. Resection should be medial to the coracoid.

30 degrees at the acromioclavicular joint obtained by rotation of the clavicle (approximately 50 degrees) and relative elongation of the coracoclavicular ligaments.

It is immediately evident that loss of motion in the acromioclavicular joint, from any cause

TABLE II

ACROMIOCLAVICULAR SEPARATION (1944–1951)

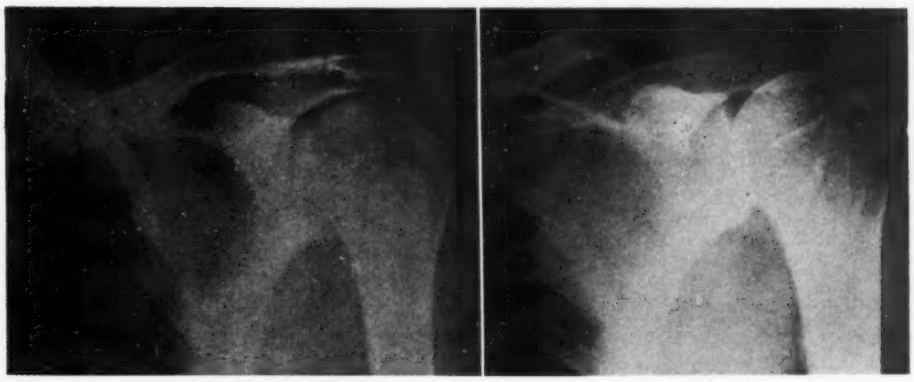
| | |
|---|---|
| Total cases treated | 76 |
| Incomplete | 32 |
| Complete | 44 |
| Conservative treatment (Watson-Jones and Urist methods) | 48 |
| Transfixation with Kirschner wires | 14 |
| Coracoclavicular screw fixation | 1 |
| Ligmentopexy | 5 |
| Partial claviculectomy | 22 |
| Cases primarily treated conservatively requiring secondary treatment | 16 |

be it dislocation, ankylosis or fixation of the clavicle to the coracoid, will manifest itself by a proportionate loss of ability to elevate the arm. Malunion of the clavicular fractures with loss of the lateral curve results in incomplete elevation by preventing effective rotation of the clavicle.

Absence of the clavicle or its outer third results in little disability.[10] Although adduction of the shoulder is permitted, no change in carrying position results. The shoulder does not sag and there is no predisposition to the scapulocostal syndrome described by Michele.[29] One does find some instability in complete elevation when supporting a weight. Here the deltoid is at a disadvantage and the clavicle tends to stabilize the shoulder. By the same token a painful joint at either end of the clavicle cries the loudest in supporting a weight with the arm over head and slight instability is usually borne with greater equanimity than pain.

We have utilized these principles as indication for resection of the outer third of the clavicle in the following conditions:

*Acromioclavicular Separation.* (Figs. 1A to D.) This very common dislocation is usually classified as complete or incomplete depending, apparently upon the degree of damage to the coracoclavicular ligaments. The majority fall into the incomplete group and conservative therapy immobilizing the clavicle in a reduced position suffices. Many become asymptomatic after a few weeks with no treatment and,



2A 2B

FIG. 2. A and B, arthritis of acromioclavicular joint with pain and limitation of motion. Resection of the outer third with complete relief of symptom; full motion restored. Postoperative film three and a half years after surgery. Note absence of regeneration.

because rotation of the clavicle is free, enjoy a full range of motion. The slight deformity at the acromioclavicular joint then has only cosmetic importance. A small proportion are complicated by a persistently painful joint and, oddly enough, in our series this disturbance is found about equally in the successfully reduced and untreated.

When the dislocation is complete, conservative therapy is rarely adequate. The joint may be reduced and transfixation accomplished by one or more Kirschner wires to maintain position until the ligaments have healed after which the wires may be removed.[12–15] This method was utilized in fourteen cases in this series and results were satisfactory in twelve. Several objections to this method of treatment are appreciated by all. The wires break,[15] they migrate,[16] elevation is limited until the wires are removed and a high incidence of traumatic arthritis results. An average disability of three months is significant from an industrial standpoint. Lastly, if the patient is seen late, there is little hope of spontaneous repair of the ruptured ligaments by this method of treatment.

Reconstruction of the coracoclavicular and acromioclavicular ligaments has been advocated by such notables as Cadenat,[17] Campos,[18] Bunnell,[19] Vargas,[20] Marble[21] and others. The operation is difficult and unless the ligaments reconstructed allow normal rotation of the clavicle some loss of motion will result. A further complication of reconstructive operations is a high incidence of painful acromioclavicular joints. In this series only two of five cases could be considered to have satisfactory results; the others required resection of the outer clavicle.

Many other methods have been suggested and include arthrodesis of the acromioclavicular joint,[23] screw fixation of the clavicle to the coracoid,[23] screw fixation of the acromioclavicular joint[25] and others. All of these would appear to prevent the normal rotation of the clavicle and thus limit shoulder motion. Such was the case with one treated in this series with the Bosworth screw. Yet Bosworth reports a follow-up study in which full motion is present in all his cases.[24]

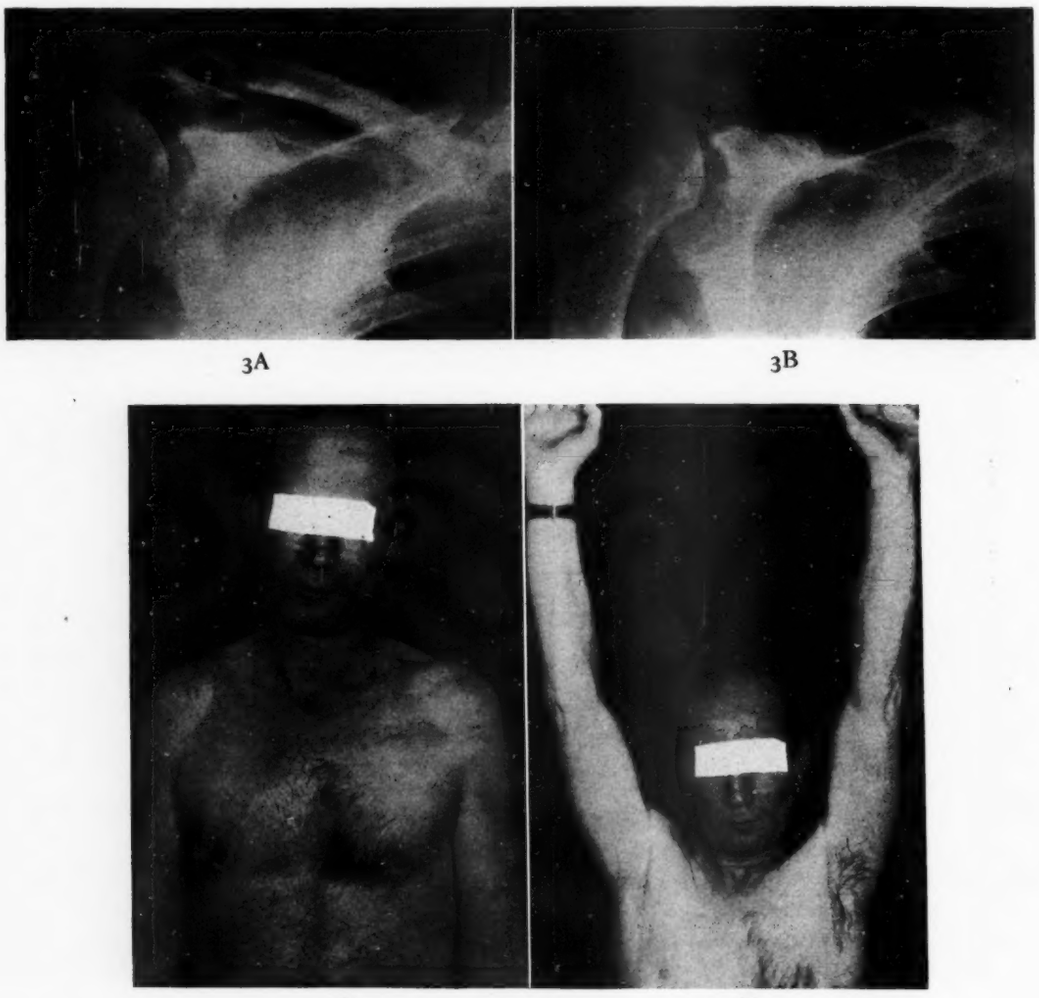
Resection of the lateral third of the clavicle is a simple approach to the completely dis-

TABLE III

RESULTS IN ACROMIOCLAVICULAR SEPARATION

| | | |
|---|---|---|
| Total cases treated | | 76 |
| Follow-up total conservative and operative | | 66 |
| Average follow-up | | 3.6 years |
| 1. Conservative therapy—primary | Total | 46 |
| Good | | 22 |
| Fair | | 8 |
| Failures (resected) | | 16 |
| 2. Operative therapy—primary | Total | 20 |
| (*a*) Transfixation with Kirschner wires | Total | 14 |
| Good | | 9 |
| Fair | | 3 |
| Failure (resected) | | 2 |
| (*b*) Coracoclavicular screw fixation | Total | 1 |
| Failure (resected) | | 1 |
| (*c*) Ligmentopexy | Total | 5 |
| Fair | | 2 |
| Failure (resected) | | 3 |
| 3. Partial claviculectomy—none primary | Total | 22 |
| Good | | 20 |
| Fair | | 2 |

Good = asymptomatic; fair = symptoms, but able to do regular work; failure = symptoms, unable to work.



3A 3B

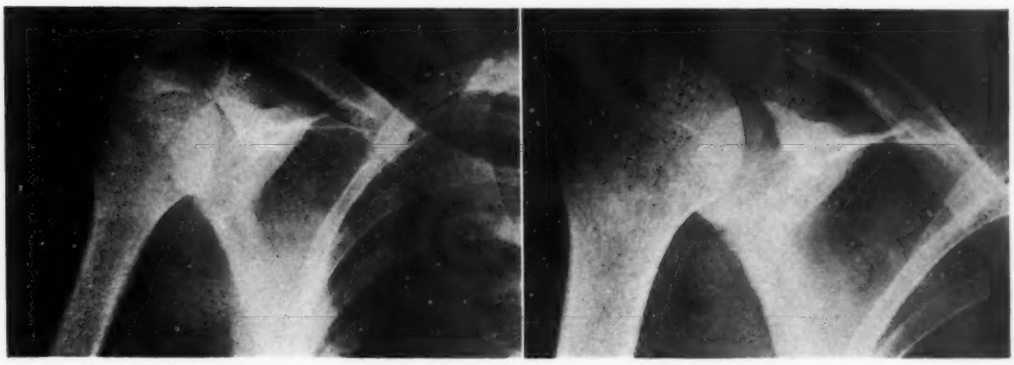
3C 3D

FIG. 3. A to D, arthritis of acromioclavicular joint following fracture of outer third of clavicle; partial claviculectomy with appearance and range of motion twelve days postoperatively.

located acromioclavicular joint. The operation is easy and the recovery rapid. The patient quickly enjoys full painless motion, and returns to work in three or four weeks or earlier depending upon his duties. If the operation is performed properly and the trapezius and deltoid sutured securely, there is no significant deformity. The clavicle must be resected medial to the coracoid or soft tissue will impinge between its lateral end and the acromion on full elevation, or the remaining attached coracoclavicular ligaments may be painful when stretched.

*Arthritis of the Acromioclavicular Joint.* (Fig. 2A and B.) This condition is relatively common in the older age group and appears to be traumatic in origin.[26,27] It occurs more frequently in the major extremity and in the laborer. It is often asymptomatic until activated by a minor injury and, when it becomes disabling, conservative treatment is of little value. Following resection of the outer third of the clavicle the patient quickly regains a full range of painless motion. Arthrodesis is tolerated apparently because the patient is already accustomed to the partial loss of elevation. However, such a procedure entails prolonged immobilization, further loss of motion and long disability. When the high failure rate of arthrodesis is added to the above, one finds little to recommend the procedure.

Other indications of partial claviculectomy include tuberculosis of the acromioclavicular joint and arthrodesis of the shoulder. Tuberculosis of the above joint is rare.[28] The disease usually has its onset in the acromion and secondarily involves the joint. The shoulder joint



4A 4B

4C 4D

FIG. 4. A to D, tuberculosis of the acromion and acromioclavicular joint; block resection; range of motion fourteen days after operation.

remains unaffected for a considerable length of time and in two cases complete block excision of the acromion and outer clavicle resulted in complete cure. As illustrated, the loss of the acromion and outer clavicle did not interfere with motion. The deltoid found firm footing on the trapezius and strength of elevation was equal grossly to the opposite side.

By excising the outer third of the clavicle one allows the scapula several additional degrees, thus the arthrodesed shoulder realizes an increased range of motion with this procedure. The additional surgery is minimal and the results gratifying.[10,11] (Figs. 3 to 5.)

### TECHNIC OF OPERATION

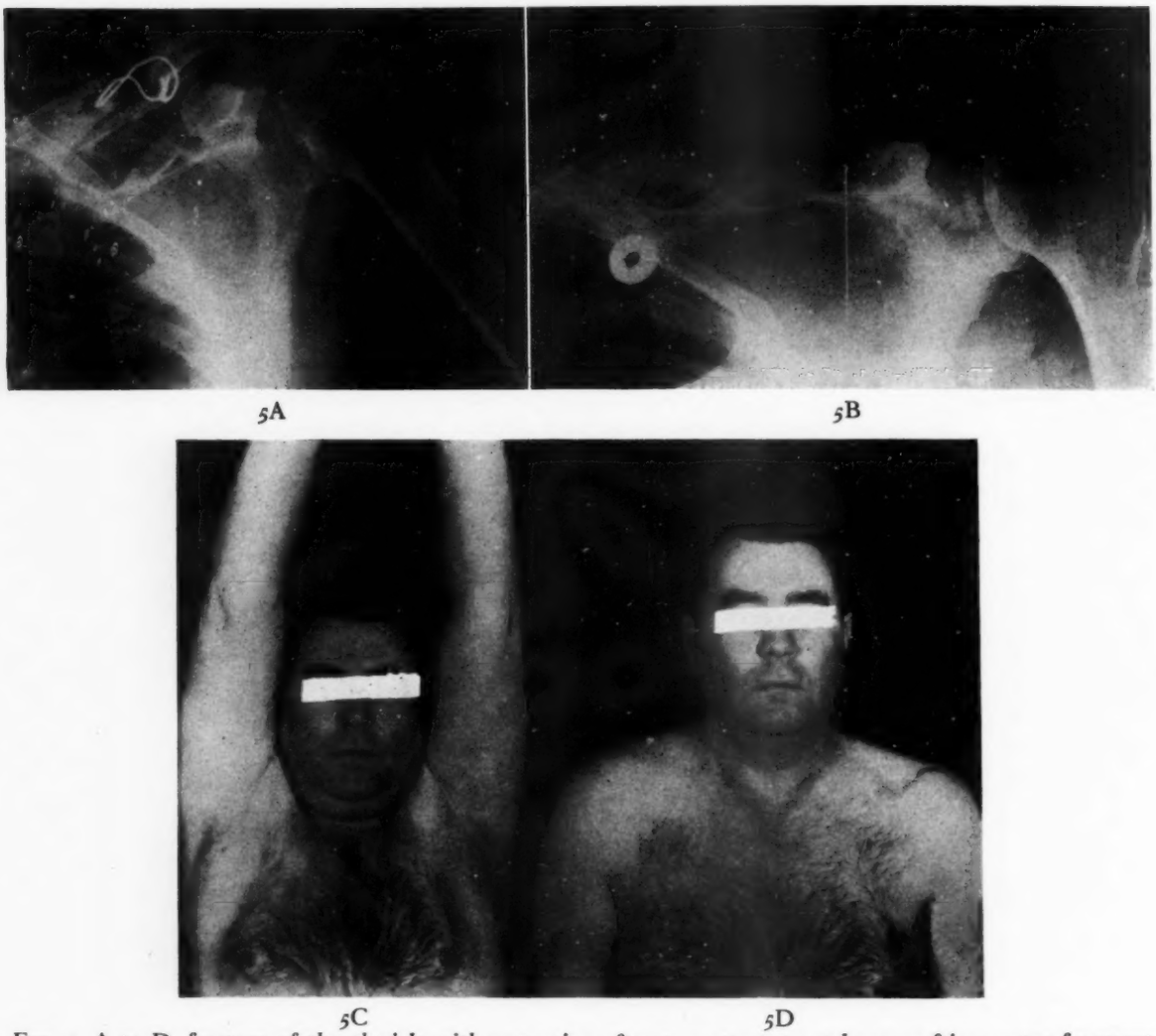
The operation may best be performed through a dorsal incision following the lateral curve of the clavicle and preserving the lateral branches of the supraclavicular nerves. The outer third of the clavicle is exposed in the interval between the attachments of the trapezius and deltoid subperiosteally. The bone is bisected just medial to the coracoid and the outer fragment removed subperiosteally. Re-

TABLE IV

OTHER RESULTS OF PARTIAL CLAVICULECTOMY

| | | |
|---|---|---|
| Arthritis | Total | 12 |
| Good | 9 | |
| Fair | 3 | |
| Tuberculosis | Total | 2 |
| Good | 2 | |
| Non-union | Total | 2 |
| Good | 1 | |
| Fair | 1 | |
| Arthrodesis, shoulder | Total | 3 |
| Good | 3 | |

Evaluation: Good = asymptomatic; fair = symptoms but able to do regular work; failure = symptoms and unable to do work.



5A 5B

5C 5D

FIG. 5. A to D, fracture of the clavicle with non-union after two attempts at bone grafting; outer fragment resected; range of motion twelve days after operation.

moval from the outer end medially is not advised as it unnecessarily disturbs the remaining clavicle and leaves its free end elevated. The wound is closed by mattress sutures between the deltoid and trapezius obliterating the periosteal bed.

We have never observed any regeneration of the outer third of the clavicle or postoperative calcification of the coracoclavicular ligaments.

The wound is dressed with elastoplast and the arm carried in a sling. Codman exercises are initiated on the fifth day and weight lifting and other exercises at two weeks. A full range of motion is present at one week in most cases.

The patient resumed work in from three to four weeks in our series, but in private practice one would expect this time to be considerably shorter.

SUMMARY

An attempt has been made to evaluate the results of partial claviculectomy in the treatment of complete separation, tuberculosis and arthritis of the acromioclavicular joint. The operation is simple, gives good functional and cosmetic results and rehabilitates the patient significantly faster than any other procedure available for the treatment of these conditions. Prolonged follow-up reveals an absence of late complications.

REFERENCES

1. MUMFORD, E. M. Acromioclavicular dislocation. *J. Bone & Joint Surg.*, 23: 799–802, 1941.
2. COPELAND, S. M. Total resection of the clavicle. *Am. J. Surg.*, 72: 280–281, 1946.
3. URIST, M. R. Complete dislocation, acromio-

clavicular joint. *J. Bone & Joint Surg.*, 28: 813–837, 1946.
4. Marottoli, O. R. Acromioclavicular separation—partial resection, clavicle. *An. de cir.*, 11: 305–308, 1946.
5. Shumacker, H. B. Resection, clavicle. *Surg., Gynec. & Obst.*, 84: 245–248, 1947.
6. Toumey, J. W. Surgery of the acromioclavicular joint. *S. Clin. North America*, 29: 905–912, 1949.
7. Ribeiro, E. B. Partial resection of the clavicle. *An. paulist. de med. e cir.*, 59: 11–12, 1950.
8. Pedemonte, P. V. Partial resection of the clavicle. *Arch. urug. de med., cir. y especialid.*, 35: 477–480, 1949.
9. Gurd, F. B. Treatment of complete dislocation, acromioclavicular joint. *Ann. Surg.*, 113: 1094–1098, 1941.
10. Inman, V. T. and Saunders, J. B. Observations on the function of the clavicle. *California Med.*, 65: 158–166, 1946.
11. Inman, V. T. et al. Observations on the function of the shoulder joint. *J. Boint. & Joint Surg.*, 26: 1–30, 1944.
12. Bohler, J. Treatment acromioclavicular separation by transarticular fixation. *Wien. med. Wchnschr.*, 100: 264–265, 1950.
13. Phemister, D. B. Treatment of dislocation acromioclavicular joint. *J. Bone & Joint Surg.*, 24: 166–168, 1942.
14. Bloom, F. A. Wire fixation, acromioclavicular separation. *J. Bone & Joint Surg.*, 27: 723–276, 1945.
15. Murray, G. Fixation of dislocations of acromioclavicular joint. *Canad. M. A. J.*, 43: 270–273, 1940.
16. Mazet, R. Migration Kirschner wires from shoulder to lung. *J. Bone & Joint Surg.*, 25: 477–483, 1943.
17. Cadenat, F. M. Treatment of fractures and dislocations, outer end, clavicle. *Internat. Clin.*, 27: 145–169, 1917.
18. Campos, O. P. Acromioclavicular dislocation. *Am. J. Surg.*, 43: 287–291, 1939.
19. Bunnell, S. Fascial graft for dislocation acromioclavicular joint. *Surg., Gynec. & Obst.*, 46: 563–564, 1928.
20. Vargas, L. Repair complete acromioclavicular dislocation. *J. Bone & Joint Surg.*, 24: 772–773, 1942.
21. Marble, H. C. Injury, Shoulder Girdle. In Bancroft and Murray's Surgical Treatment of the Motor Skeletal System, part II, p. 289. Philadelphia, 1945. J. B. Lippincott.
22. Caldwell, G. D. Arthrodesis, acromioclavicular joint. *J. Bone & Joint Surg.*, 25: 368–374, 1943.
23. Bosworth, B. M. Acromioclavicular separation. *Surg., Gynec. & Obst.*, 73: 866–871, 1941.
24. Bosworth, B. M. Acromioclavicular separation. *Ann. Surg.*, 127: 98–111, 1948.
25. Tyler, G. T. Acromioclavicular separation. *Am. J. Surg.*, 58: 245–247, 1942.
26. Oppenheimer, A. Arthritis, acromioclavicular joint. *J. Bone & Joint Surg.*, 25: 867–870, 1943.
27. Oppenheimer, A. Lesions of the acromioclavicular joint causing pain and disability. *Am. J. Roentgenol.*, 51: 699–706, 1944.
28. Corci, G. Acromioclavicular joint tuberculosis. *Ortop. e traumatol.*, 17: 301–308, 1949.
29. Michele, A. A. Scapulocostal syndrome. *New York State J. Med.*, 50: 1353–1356, 1950.

# EPIPHYSEAL INJURIES OF THE RADIAL HEAD AND NECK*

SAWNIE R. GASTON, M.D., FREDERICK M. SMITH, M.D. AND ORREN D. BAAB, M.D.
*New York, New York* *Chicago, Illinois*

INJURY to the radial head and neck is of less frequent occurrence in children than in adults. During the fourteen-year period from 1938 to 1952 there were 479 cases of injury to the radial head and neck in all age groups treated on the Fracture Service of the New York Orthopedic—Columbia-Presbyterian Medical Center, New York City, of which 100 occurred in children with open epiphyses, a percentage of 21. Jeffrey[1] reported eighty such cases in a total of 450 injuries to the radial head, or 18 per cent. Of the 100 cases studied in this series seventy-four were adequately documented to report. They are presented in four groups according to their pathologic classification.

## PATHOLOGIC CLASSIFICATION

*Group I.* This group, consisting of twenty-two cases, includes injuries to the upper radial epiphysis disgnosed on clinical grounds alone. Children who had sustained a fall on the outstretched hand or forearm and complained of elbow pain and showed tenderness centered at the upper radial epiphysis for as long as a week, with painful limited elbow motion and hemarthrosis of the elbow joint, despite a negative x-ray, were considered to have injured the radial epiphysis and not to have just "sprained" the elbow. Treatment consisted of a protective splint or sling for seven to twenty-one days until epiphyseal tenderness subsided, followed by progressive active use of the extremity. All had full motion at twenty-two days. They were followed an average of two years seven months without clinical or x-ray evidence of growth disturbance.

*Group II.* The thirty-nine cases in this group includes those patients with x-ray evidence of fracture of the radial head and neck, but with minimal or no displacement. The most frequent x-ray appearance was a 10 degree tilt of the radial epiphysis with buckling of the cortex of the neck. Others showed a transverse fracture of the neck without tilt or displacement and one showed a slight lateral shift of the epiphyseal plate carrying a triangular fragment of the metaphysis with it. Two showed a tilt of 15 degrees. They were followed an average of three years and all had normal anatomy, full function and full use. None had clinical evidence of growth disturbance, either locally or in radial length. The two patients with a 15 degree tilt showed premature epiphyseal closure and some widening by x-ray. The one with a lateral shift of the epiphyseal plate also showed widening of the radial head by x-ray.

*Group III.* This group, consisting of six cases, includes fracture of the proximal radius with a 20 to 70 degree tilt of the radial epiphysis. Three patients had no reduction. They were followed an average of three years and all showed some limitation of elbow motion. X-ray showed enlargement of the radial head and premature epiphyseal closure. All had normal radial length. Their ages were eight, nine and eleven years. The remaining three had an excellent reduction, two by operation because of associated fracture of the ulna and one by closed reduction. They were followed an average of three and a half years and all had normal anatomy, full function and full use. X-ray showed slight widening of the radial neck and normal radial length. Their ages were eight, twelve and thirteen. (Figs. 1 and 2.)

*Group IV.* The seven cases in this group includes patients with fractures of the radial epiphysis associated with a dislocation at the elbow. All showed a complete avulsion with displacement and rotation of the radial epiphysis. It is unlikely that complete avulsion of the epiphysis occurs without dislocation.

## CASE REPORTS

### *Group IV*

CASE 1. This eight year old child, injured two days prior to admission, showed a posteromedial

* From the Department of Orthopaedic Surgery, College of Physicians and Surgeons, and the Fracture Service, New York Orthopaedic Hospital, Columbia-Presbyterian Medical Center, N. Y.

1A 1B 1C

FIG. 1. Group III, closed reduction. A, a 30-degree tilt of the radial epiphysis. B, two years following closed reduction; slight widening of radial head; normal radial length. Full function. C, normal opposite elbow.

dislocation of both bones at the elbow with an avulsion of the olecranon apophysis and complete avulsion and rotation of the radial epiphysis. A reduction of the dislocation had been performed elsewhere, but dislocation had recurred. Open reduction was decided upon, but had to be postponed three days because of bleb formation. A double operative approach was employed: Posteriorly to wire the olecranon apophysis and anteriorly to replace the radial head. Immobilization in a posterior splint was continued for five weeks. The patient had a dorsal interosseous nerve palsy postoperatively which cleared in three months only to recur again in four years and become permanent because of attrition due to an encircling suture. As early as five months postoperatively there was evidence of a proximal radioulnar synostosis which consolidated with the years. The patient was followed six years. Elbow flexion and extension were complete, but forearm rotation was frozen at 20 degrees supination. (Fig. 3.)

This result was due to unavoidable delay in operating and too much operating. It created a memory in the minds of the professional staff which has led to subsequent operative decisions which are now open to challenge. The fear of synostosis as a result of open reduction to replace a displaced radial epiphysis influenced in part the decision to remove the radial head in the two cases next reported.

CASE II. This seven year old child, admitted the day of injury, showed a modified Monteggia type of fracture with fracture and anterior angulation of the proximal ulna shaft and a forward dislocation of the radius with complete avulsion of the radial epiphysis. The ulna fracture had an open wound. Immediate operation was performed. The wound was excised and cleansed and the ulnar fragments secured with plate and screws through a Boyd[2] approach. An attempt was made to replace the radial head, but it would not stay in position and moved eccentrically. For this reason, and for fear of synostosis, it was removed. Early motion, with splint protection for five weeks, constituted the postoperative management. The patient was followed eight years and had full motion and full use without symptoms. There was, however, a 10 degree increase in carrying angle and a 5 degree lateral instability of the elbow.

By x-ray measurement the injured radius was 3 cm. shorter than its opposite fellow at time of epiphyseal closure. The ulna was 1.5 cm. shorter than normal, indicating a certain compensatory retardation of growth. At the wrist the radial styloid was 1.5 cm. shorter than the ulna styloid, compared with the normal relationship of the opposite side. (Fig. 4.)

CASE III. This ten year old child was injured nine weeks prior to admission and operation. She showed a complete avulsion and rotation of the radial head and had had a previous attempt at

FIG. 2A. Group III, open reduction. A 70-degree tilt of the radial epiphysis and fracture separation of the olecranon.

2B 2C

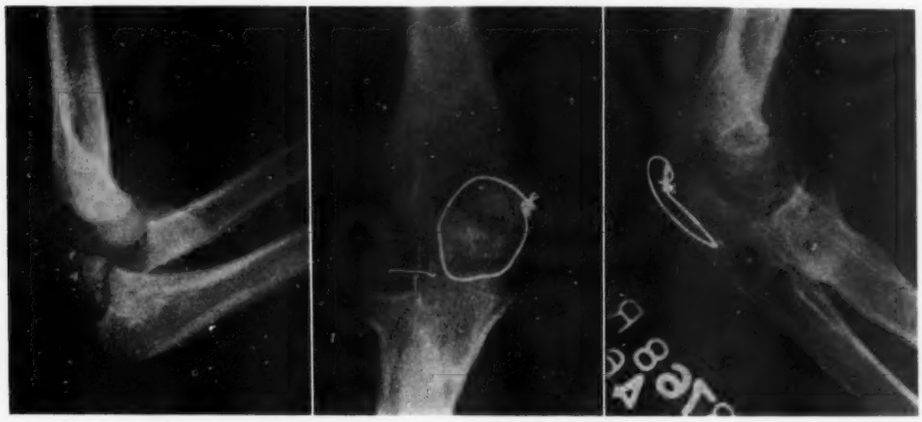
FIG. 2. Group III, open reduction. Four and a half years following open reduction; B, slight widening of radial head; normal radial length. Full function. C, normal opposite elbow.

reduction by closed methods five days following injury. There was no absolute record of dislocation, but it is assumed. Excision of the radial epiphysis was decided upon preoperatively because of the interval since injury with almost certain growth disturbance regardless of replacement and for fear of synostosis. This rationale will be discussed later. Active motion was started two days following removal of the radial epiphysis. The patient has been followed over four years. She has crepitus and clicking in the elbow with motion. Eating with a fork is difficult because of lack of supination and writing is uncomfortable. She lacks 20 degrees of full flexion and 80 degrees of full extension. Pronation is full, but supination is restricted at 15 degrees. X-rays show a bizarre regrowth of the proximal radius which has excavated the capitellum.

By x-ray measurement the injured radius was 1.2 cm. shorter than its fellow at time of epiphyseal closure. The ulna was 0.7 cm. shorter than normal. At the wrist the injured radius was only 0.5 cm. short. (Fig. 5.)

CASE IV. This seven year old child, admitted the day of injury, showed a posteromedial dislocation of both bones at the elbow, a displaced fracture of the olecranon and complete avulsion of the radial epiphysis. Immediate open reduction was performed following two unsuccessful attempts to replace the radial epiphysis by manipulation. The radial epiphysis was found to be rotated 180 degrees with its articular surface pointing distally and it was wedged between the radial neck and capitellum. The joint capsule was torn and the orbicular ligament completely disrupted.

A posterolateral approach was used and it was found that the radial epiphysis had no soft part



3A 3B

FIG. 3. Group IV, Case I. A, complete avulsion of radial epiphysis and displacement of olecranon apophysis. B, three and a half years postoperatively showing radio-ulnar synostosis.

4A 4B 4C 4D

FIG. 4. Group IV, Case II. A, Monteggia type fracture with complete avulsion of radial epiphysis. B, Postoperative x-ray showing reduction and fixation of ulnar fracture and excised radial head. C, seven years following operation with 1.5 cm. difference in styloid levels at the wrist. Full function. D, normal opposite forearm.

attachments to hold a suture. Consequently, it was impacted in anatomic position with an elevator, following which it moved with the radius without displacement. A posterior moulded splint was applied and active motion started on the tenth day. X-rays one week later showed some loss of position of the radial epiphysis. The patient was followed six years. There was a palpably enlarged radial head, a 5 degree increase in carrying angle, a 10 degree loss of full flexion and considerable restriction of rotation with pronation zero and supination only 15 degrees. X-ray at three years showed an enlarged eccentric radial head and premature closure of the epiphysis. There was no disturbance in radial length. Radial head resection was refused. (Fig. 6.)

CASE V. This nine year old child, admitted the day of injury, showed complete avulsion of the radial epiphysis. Immediate open reduction was performed through a posterolateral approach. It is of interest that whereas the preoperative clinical examination and x-ray did not show a dislocation, there was a large stellate laceration of the lateral capsule found at operation, indicating that dislocation had occurred, but had reduced spontaneously. The radial epiphysis was rotated so that its articular surface faced anteromedially. It was replaced and impacted with an elevator and

5A 5B 5C 5D

FIG. 5. Group IV, Case III. A, complete avulsion of radial epiphysis. B, four years following excision of radial epiphysis showing regrowth of radius from epiphyseal remnant and excavation of capitellum. C, four years following operation with only 0.5 cm. difference in styloid levels at the wrist. Limited function due to intra-articular damage. D, normal opposite forearm.

6A 6B 6C

FIG. 6. Group IV, Case IV. A, complete avulsion of radial epiphysis with displaced fracture of olecranon and dislocation. B, ten days following open reduction. C, three weeks following operation showing loss of position of radial epiphysis.

remained firmly in place without fixation. The elbow was immobilized for eleven days only. At seven months the patient had full motion and full use. A letter at two years described full function and no symptoms. X-ray at seven months showed merely slight widening of the neck. (Fig. 7.)

CASE VI. This twelve year old child, admitted the day of injury, showed a medial subluxation of the radius and complete separation and lateral displacement of the radial head. Closed reduction was unsuccessful and immediate operation was performed. The orbicular ligament was torn and found interposed between the fragments. The radial head was replaced but not impacted. Subsequent x-rays showed loss of position despite immobilization for one month. The patient was followed two years. Flexion lacked 20 degrees, extension was full, pronation was 70 degrees and supination was full. There was a 10 degree increase in carrying angle. X-ray showed a non-union of the radial head. It is of interest that the radial epiphysis of the opposite side was closed normally six months following operation at the age of twelve and a half years. (Fig. 8.)

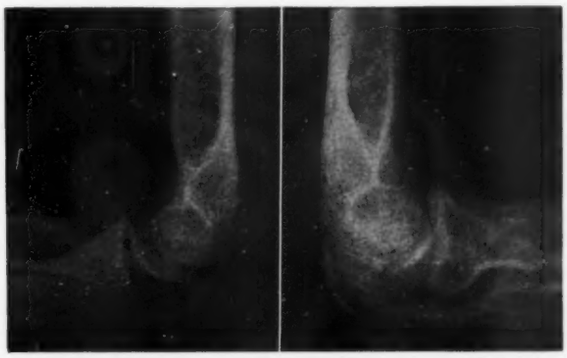
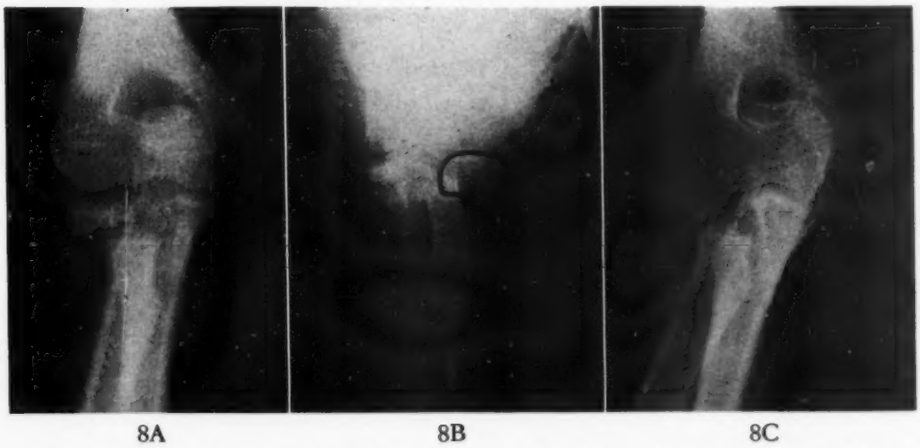


FIG. 6D. Group IV, Case IV. Three years following operation showing enlargement of radial head compared to opposite normal; limited function.

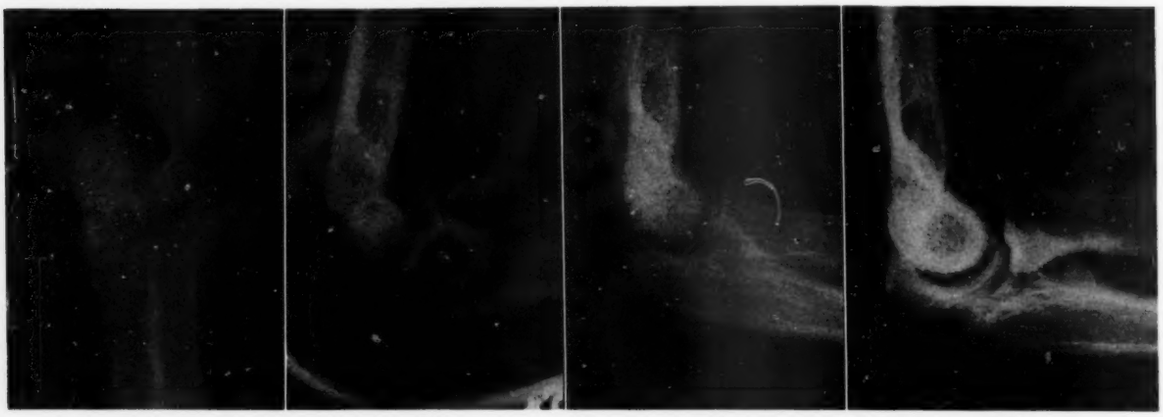
7A 7B 7C

FIG. 7. Group IV, Case V. A, complete avulsion of radial epiphysis. At operation a tear was found in the capsule, indicating a spontaneously reduced dislocation. B, seven months following operation and replacement of radial epiphysis showing minor thickening of radial head. Full function. C, normal opposite elbow.



8A 8B 8C

FIG. 8. Group IV, Case VI. A, lateral displacement of radial epiphysis with interposition of orbicular ligament. Note separation of fragments. B, ten days following open reduction. Loss of position is sketched in black over faint x-ray outline. C, two years following open reduction showing non-union of radial head. Limited function.



9A 9B 9C 9D

FIG. 9. Group IV, Case VII. A, complete avulsion of radial epiphysis with dislocation. B, anatomic replacement following open reduction. C, loss of position of radial epiphysis four weeks postoperatively. D, two and a half years postoperatively showing enlargement of radial head. Limited function.

CASE VII. This thirteen year old patient, admitted the day of injury, showed a posterior dislocation of both bones at the elbow with complete displacement and 90 degrees rotation of the radial epiphysis. An immediate open reduction was performed through a lateral approach. The orbicular ligament was found to be completely torn. A sling of periosteum was still attached to the radial neck and displaced fragment. The head was replaced, but not impacted. Although a posterior moulded splint was worn for one month following operation, it was removed at seventeen days to test the range of elbow motion. Subsequent x-ray showed loss of position of the radial epiphysis. Two and a half years following injury examination showed an enlarged, irregular radial head. Flexion and extension were full, but rotation was limited with pronation to 15 degrees and supination to 80 degrees. Radial head resection was performed and full motion obtained. (Fig. 9.)

## COMMENTS

Of the seventy-four patients 82 per cent may be considered to have had a minor injury which required symptomatic treatment only. The remaining 18 per cent, or thirteen patients, requiring more energetic treatment were surprisingly few in number, approximately one a year for the fourteen-year study. The more serious injuries classified as group IV numbered only seven patients. It must be appreciated that such a limited clinical experience necessarily circumscribes discussion of the proper management of these injuries. This end result study, however, has afforded information which indicates the effect of trauma of varying degree to the proximal radial epiphysis and the result of this influence on the subsequent anatomy and function of the involved structures in these few cases. These factors are discussed under the following three headings: (1) factors which cause restriction of elbow motion; (2) factors which determine alteration in radial length; (3) factors which emphasize the importance of the time element in treatment.

### FACTORS WHICH CAUSE RESTRICTION OF ELBOW MOTION

Follow-up x-rays of patients with a slight lateral displacement or a 15 degree tilt or less of the radial epiphysis showed only slight widening of the radial head and neck insufficient to interfere with elbow joint function. These patients did not require a reduction and all obtained full function without symptoms.

X-rays of patients with an uncorrected tilt of 20 degrees or more, or those with a recurrence of deformity due to loss of position following reduction, showed enlargement and irregularity of the radial head inconsistent with normal function. This was due to subperiosteal new bone formation and alteration in local growth of the radial epiphysis which together produced an overgrown and eccentric radial head. (Fig. 10.) Reduction and maintenance of reduction is mandatory in patients in group III with a tilt of 20 degrees or more and in group IV with complete displacement of the radial epiphysis. Three patients in group III and one in group IV had an anatomic and well maintained reduction and enjoyed full elbow motion. Three patients in group IV lost position following reduction and enlargement and

irregularity of the radial head developed which was sufficient to interfere with function. It is strongly urged that the radial epiphysis be impacted or stabilized in position at time of operation and that external plaster of Paris immobilization be continued uninterrupted for a minimum of four weeks postoperatively.

Closed reduction is considered preferable for Group III cases when not complicated by associated injuries. An excellent method has been described by Patterson[3] and again by Goldenberg.[4] Open reduction is required generally for group IV cases and it is recommended that a posterolateral elbow approach be used for displacement of the radial epiphysis as a single injury and the Boyd approach for combined access to radius and ulna in associated injuries.

### FACTORS WHICH DETERMINE ALTERATION IN RADIAL LENGTH

Seventy-four cases, including all degrees of injury to the proximal radius, have been presented and only those two in which the radial epiphysis was excised showed any growth disturbance in radial length. Many patients showed premature closure of the epiphysis, but by clinical and x-ray follow-up measurements the injured radius was equal in length to the opposite side. The radial epiphysis survived in all instances of replacement in this series and none of these patients showed a disturbance in radial length. Key[5] reported replacement at forty-eight hours following injury with survival and an excellent clinical result. Both patients in whom the radial epiphysis was excised showed a compensatory retardation of growth in the length of the ulna and a clinically insignificant discrepancy in styloid levels at the wrist. The result in one was excellent with full motion and full use eight years after operation. This patient was a girl seven years of age at the time of radial head removal and the radial styloid was 1.5 cm. shorter than the ulna styloid at age fifteen. The opposite proximal radial epiphysis closed at age twelve. The other had a poor result, not because of radial shortening but because of a bizarre regrowth of the proximal radius due to incomplete removal of the epiphyseal cartilage and subsequent intra-articular damage. This patient was a girl ten years of age at time of excision, and at age fourteen showed only 0.5 cm. difference in styloid levels at the wrist. The opposite proximal radial epiphysis closed at fourteen.

FIG. 10. Subperiosteal new bone formation at four weeks along radial neck on side of displacement follows incomplete reduction or loss of position. This new bone formation is the precursor of an enlarged radial head and limited function.

Excision of the radial epiphysis has been condemned for years and yet if one may judge from these two patients, it may be a useful alternate procedure at the proper age. Should any difficulty be encountered in replacing or securing a displaced radial epiphysis in a girl of twelve or a boy of fourteen years or older, it would seem justifiable to remove the radial head provided the entire epiphyseal cartilage and neck of radius also are excised.

### THE TIME FACTOR IN TREATMENT

Proximal radio-ulnar synostosis occurred as a complication in Case 1, group IV, in this series. This patient was operated upon five days following injury through two approaches; one posteriorly to wire a displaced olecranon apophysis and one anteriorly to replace the radial head. Synostosis had occurred in two other patients previous to the period of this study. One was operated upon seven days following injury, and the radial epiphysis secured by a suture passed through a drill hole in the radial neck. The other was operated upon forty days following injury, and periosteum and callus were removed from the radial neck and 2.5 cm. of proximal radius excised. The factor of late operation and undue operative trauma was common to all three cases.

It is believed that the following three points are of major contributory significance in the production of proximal radio-ulnar synostosis in late operation: (1) The presence of callus which is stimulated to overgrowth by the trauma of operation; (2) operative insult to the adjacent ulna providing a recipient area for cross union; (3) the intimate anatomic juxtaposition of proximal radius and ulna.

To prevent this complication, operation later than forty-eight hours after injury should be avoided. Denied this choice, recent developments suggest that the use of cortisone,[6] hyaluronidase and oxycel gauze may be useful adjuncts to late operation. There were eight patients operated upon within twenty-four hours of injury in this series and all avoided this complication.

## CONCLUSIONS

Seventy-four cases of injury to the proximal radial epiphysis are presented in four groups according to their pathologic classification.

Group I, diagnosed on clinical grounds and differentiated from the so-called "sprained elbow," resulted in full function and no growth disturbance.

Group II, with x-ray evidence of fracture and a tilt of the radial epiphysis of 15 degrees or less, obtained full function without reduction.

Group III, with a tilt of 20 degrees or more, resulted in excellent function following successful reduction. Reduction is obligatory in this group.

Group IV, cases with dislocation, complete displacement of the radial epiphysis and associated injuries, are presented individually. Complications and recommendations for treatment are discussed.

## REFERENCES

1. Jeffrey, C. C. Fractures of the head of the radius in children. *J. Bone & Joint Surg.*, 32-B: 314, 1950.
2. Boyd, H. B. Surgical exposure of the ulna and proximal third of the radius through one incision. *Surg., Gynec. & Obst.*, 71: 86, 1940.
3. Patterson, R. F. Treatment of displaced transverse fractures of the neck of the radius in children. *J. Bone & Joint Surg.*, 16: 695, 1934.
4. Goldenberg, R. R. Closed manipulation for the reduction of fractures of the neck of the radius in children. *J. Bone & Joint Surg.*, 27: 267, 1945.
5. Key, J. A. Survival of the head of the radius in a child after removal and replacement. *J. Bone & Joint Surg.*, 29: 148, 1946.
6. Blunt, J. W. et al. Effect of cortisone on experimental fractures in the rabbit. *Proc. Soc. Exper. Biol. & Med.*, 73: 678, 1950.
7. Gaston, S. R., Smith, F. M. and Baab, O. D. Adult injuries of the radial head and neck. *Am. J. Surg.*, 78: 631, 1949.

## DISCUSSION OF PAPERS BY DRS. NEER, BROWN, AND MCLAUGHLIN, WAGNER, AND GASTON, SMITH AND BAAB

Gordon Mackay Morrison (Boston, Mass.): To take up briefly Dr. Neer's paper, two months ago I did one of these Neer prosthetic operations on a shoulder at the Boston City Hospital. After inserting the prosthesis I found that it was a little loose. There is, of course, a hole in the neck below the head partially down the stem through which you are supposed to thread some wire, as I understand it, and through the cortex of the bone, to prevent rotation. I am wondering if varying sizes of this prosthesis with a little broader and narrower stem to hold it firmer so that it does not tend to rotate would be helpful. Perhaps Dr. Neer can tell us what his ideas are on that.

As to Dr. Wagner's paper the first time I saw parts of the clavicle taken out were those shown by the late Fraser Gurd in Montreal. He showed some thirty-odd cases one morning, and I have never seen such magnificant results in my life. Removal of head, fusion, the other operative procedures, in our hands, have not proved to give us much of an end result, and I am just wondering if we should not pay more strict attention to this removal theory, because it certainly seems to be pretty good.

Dr. Gaston, I would like to inquire, in your first case, how you ever got that radial head manipulated back so perfectly. I would like to have you tell us exactly how you did it.

Jere W. Lord, Jr. (New York, N. Y.): I would like to focus my remarks on the paper by Dr. Wagner. He has certainly done some exceedingly fine work in this field. In a selected group of patients with arterial obstruction, intermittent or complete, we carried out partial claviculectomy of the middle half. In one case we were not as successful as Dr. Wagner in preventing regeneration. Regeneration took place and the same syndrome recurred five months later. Another patient did not have regeneration in a period of four months, but the ends of the clavicle fell closer together as a result of the resection. When this man thrust his shoulders forward, he experienced a shooting pain in his arms and hands, as though the ends of the fragment were pressing on the brachial plexus. Later we resected the remaining segments. After a year there has been no regeneration of either clavicle. Function is good and the patient is able to work. An x-ray taken some six months after total claviculectomy and removal of the periosteum

showed signs of regrowth. The patient does heavy work but does experience fatigue in the pectoral muscle region. Other than that his arms are free of any circulatory difficulty.

Another patient had bilateral total claviculectomy in two stages. The cosmetic result is not too bad and the patient has normal function and normal circulation in her hands. She is able to do everything without any muscle discomfort.

Of nine patients (thirteen limbs) who had been operated upon for vascular symptoms, the results were good in all but one case which was extremely disappointing. In three with arterial thrombosis the results were also good.

We have only considered this operation in a highly selected group of patients who had had medical measures carried out for a sufficient length of time to rule out the possibility of returning to normal function and normal work. In that small group with arterial insufficiency due to intermittent or permanent blockage of the subclavian artery, it seems to be worth while.

FREDERICK M. SMITH (New York, N. Y.:) Apropos of radial head fractures in children, I have been particularly interested, along with Dr. Gaston, in reviewing these. Just to refresh my memory on what Sir Reginald Watson-Jones had to say on the subject, I looked over his writings.

He mentions only two types of radial head fractures, first the greenstick fracture of the neck, and second the tilt of the radial head, which would correspond to group III that Dr. Gaston described. Dr. Watson-Jones did not mention the type of radial head fracture in which the entire radial head epiphysis is displaced in a lateral or posterior direction, and usually rotated 90 degrees from its normal axis. He does recommend closed manipulation for those with tilt and, if this is unsuccessful, operation.

One thing he does say, which I heartily disagree with, is that you should not operate upon these patients early. He advises operating somewhere around seven to ten days following injury. I personally believe that this is the worst possible time to operate in these cases. Two patients in this series of three cases of synostosis that Dr. Gaston mentioned were operated upon somewhere around six to seven or eight days. As a matter of fact, all synostosis in these children have come from cases which were not operated upon immediately. We prefer an immediate operation.

In all the radial head fractures with marked tilt, we have not had a single medial epicondyle avulsed, and in four or five of them we had olecranon fractures of the type Dr. Gaston showed you.

As far as the radial head excision is concerned in these children, I frankly admit that I am responsible for both of the cases that he mentioned. I did it because of fear of possible synostosis, but at that time, I had not realized that those synostosis cases had all been operated upon at a late date.

One of the patients whose radial head I resected was seen in follow-up clinic recently. She is now fourteen years old, has complete range of function, and the growth disturbance she formerly had at the wrist is vaguely discernible clinically. She has a full range of motion and full strength.

The other one that overgrew the radius was a case that came to me three weeks after injury. The radial head was displaced into the posterior lateral compartment and operation was delayed still further. Believing that three weeks was the wrong time to operate I operated at nine and a half weeks and just incised the capsule, plucked out the radial head and did nothing further. In about two years the patient began to show evidence of the upper end of the stump increasing in length. I had not touched this at operation. The epiphyseal cartilage must have been left on it, continued to grow, and a cavity appeared in the capitellum. Recently, I removed the upper end of that radial neck which was surrounded with fibrous tissue and some pseudosynovial membrane. I do not think I improved the flexion or extension which was limited to about 55 to 100 degrees.

BRADLEY L. COLEY (New York, N. Y.): I should like to confine my discussion to Dr. Wagner's interesting paper, and at the same time suggest another indication for either partial or incomplete claviculectomy, namely, for certain benign tumors of bone and for carefully selected tumors of low grade malignancy together with a group of allied conditions which are not actually neoplasms.

We have found that certain osteochondromas, for example, are histologically benign but have a definite tendency to become malignant in later years. In one case carefully followed, this transition took place seventeen years after the patient was first seen. We therefore take a rather aggressive attitude toward osteochondroma and when this lesion occurs in the clavicle, we regard claviculectomy as indicated.

I should like to add that I was very much impressed with Dr. Lord's experience with total removal of the clavicle. I recall a case that I saw during the war at one of the Army general hospitals. The patient was a soldier who had a peculiar lesion involving the entire clavicle which on x-rays resembled a sclerosing osteitis. Since this was a disabling condition, the Chief of Orthopedic Surgery at this hospital performed a complete removal of the clavicle. I saw the patient just prior to his discharge when he had a full range of motion, and I later learned that he was able to resume full duty as an infantry soldier.

ARTHUR R. METZ (Chicago, Ill.): I would like to ask Dr. Neer the position of the arm following the operative procedure, as to whether he dresses it

down to the side, or elevates it, and if elevated, how high?

It has been our experience in injuries around the shoulders, where there has been a great deal of hemorrhage into the tissues, to suspend the arm, elevated above the head, with the patient in bed. We make this a routine practice, whether operative or not. The arm is elevated as early as possible and the patient kept in bed two to three weeks until absorption takes place and passive and active motion used. The patient always gets the arm down, but sometimes when dressed down to the side, he does not get it up. We believe that it is time well spent in order to preserve useful motion. I would like to have Dr. Neer discuss his postoperative treatment.

CHARLES S. NEER (closing): In reply to the question from the Chair the postoperative treatment of these patients included varying periods in which the arm was maintained in the abducted position following head resection. Some arms were dressed at the side immediately after the operation. At least half of them were maintained in abduction as long as three weeks, and some of them as long as thirty-five days. Maintenance of abduction for three weeks postoperatively might give a better result at six months, but the eventual result, I think, would not be improved. During the initial postoperative period the glenohumeral joint is flail. Later fibrous and osseous tissues stabilize the joint and the late follow-up result is the same regardless of the immediate postoperative treatment, whether it be pendulum exercises or splinting in abduction.

In reply to Dr. Gordon Morrison's question regarding the looseness of the prosthesis which he used, as he well knows, and I would like to emphasize again, this prosthesis is very much in the experimental and developmental phase. I believe we will need at least three sizes. However, regardless of present technical faults, I think the idea of supplying a fulcrum in the upper humerus to supplant the lost head, particularly in injuries in which the head has no blood supply, is very sound. This non-weightbearing joint in which motion is the principal function is particularly well suited for replacement. I think a prosthesis could be expected to hold up better here than in the hip.

I might add one other remark and that is another indication for Dr. Wagner's excision of the outer end of the clavicle, based on the one case in which the outer 1 inch of the clavicle was removed with humeral head resection. The principle is as in an arthrodesis. This particular patient gained more motion than the other resection cases.

CARRUTH J. WAGNER (closing): I wish to close with just a word of caution. The resection of the outer third of the clavicle for acromioclavicular separations gives such better immediate results than the more standard and better evaluated methods—there is a tendency to include more and more cases into the group of primary resections. Until the resection of the outer third of the clavicle has been evaluated for a twenty- or thirty-year period to see what the life history of the condition is, I think that it should be reserved for those separations that fail to succumb to more standardized methods of treatment.

SAWNIE R. GASTON (closing): In answer to Dr. Gordon Morrison's question, how did I get that radial head back, I must confess I did not. One of the nice things about belonging to a service like the Fracture Service is that simulated experience accrues by inheritance. I do not remember which member of the staff reduced that radial head; I agree it was a beautiful job.

It might be worth mentioning a method of closed reduction which has been described by Dr. Paterson and again by Dr. Goldenberg. They point out that if traction is applied to the extended elbow with the forearm in supination and forced adduction, to open up the lateral joint space, the radial epiphysis will be levered into position by the action of the taut lateral collateral ligament and its insertion into an intact orbicular ligament. This manipulation should be augmented by local digital pressure over the radial epiphysis.

# REFRACTURES IN CHILDREN

WILLIAM H. CASSEBAUM, M.D. AND RICHARD B. HAMILTON, M.D.

*New York, New York*

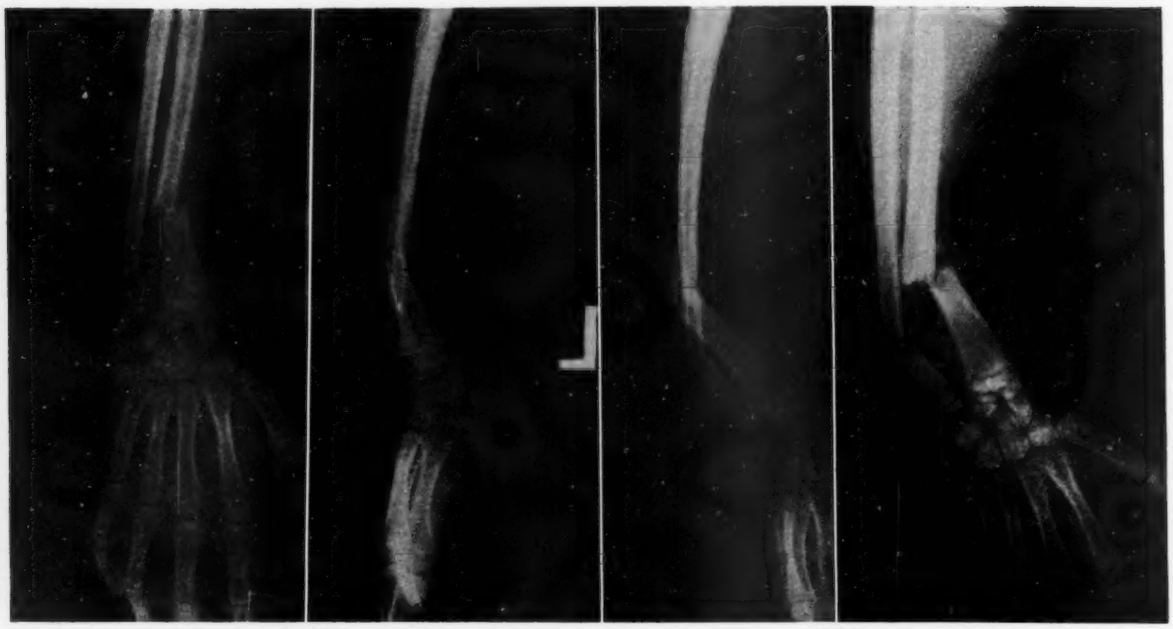
FOR some years we have been interested in and have collected cases of refracture of bones at the same location. To us this has meant the location of the refracture was determined by the trauma being identical; that the bones had an anatomic site of weakness; or that there remained a point or points of weakness after healing. In most instances it is the latter assumption that is correct.

Happily, refracture is not a common occurrence. Although Buttner quotes Kepel as finding nine in sixty-six forearm fractures in children and Bruner as finding seven in thirty-four. The incidence, therefore, in these series would approximate 14 per cent and 20 per cent. Buttner had 14 in 238, or 6 per cent. We cannot state with accuracy our own incidence of refracture in the forearm bones but we have collected eighteen cases while caring for some 280 forearm fractures, making an approximate incidence of 6 per cent also. As the report of refractures adds little to the prestige of a clinic, and human nature being what it is, I am sure the larger series are not published, perhaps not even reckoned. In the interest of preventing the avoidable refractures we present this series.

As we will presently show the great majority of refractures occur in the forearm of children. To prevent some of these we would have to change the nature of children. That we cannot do this should give us no chagrin; but on the other hand if we knew more about the strength of bony union, many refractures would be eliminated. Practically, by admonishing our patients to be cautious, we avoid refracture in older patients who retain unpleasant memories of their injury, but children soon forget and run and play with the same abandon as before. Our knowledge of the strength of their bony union is quite inexact. We know more about it in the tibia of the rat but this knowledge cannot be translated in human terms. If we are quite honest about it, there are many things about the healing of fractures which we do not know. Only a few years ago we were debating whether or not periosteum produced callus. Today there are still those who say a little displacement makes for more callus and stronger union. Some add that a little motion at the fracture site stimulates callus formation. Although Eggers has devised ingenious experiments on the value of compression, he has not convinced Watson-Jones who believes it is the immobilization that is important. Bohler on the other hand believes shortening of long bones is friendly to healing and distraction the great deterrent. In the light of this confusion our lack of knowledge about the strength of union is understandable, regrettable as it may be.

That certain fractures in the forearm of children are slow to heal has been known by many for a long time. Dr. Herbert Bergamini twenty-five years ago taught that middle third fractures in children should be splinted twelve to fourteen weeks or bowing or refracture might occur. Watson-Jones says, "Fractures of the radius and ulna are usually united in 6 weeks," but he adds, "When there is a fracture at the junction of the middle and lower third of the ulna it is unsafe to remove the plaster until there is radiographic evidence of union." The crux of the matter is what constitutes radiographic evidence of union, what is the strength of the union by "periosteal" callus. How strong is the union when callus is present on the concave side and absent on the convex? What is the significance of finding the bone ends still discernible in a sheath of "periosteal" callus?

In this group of children there were twenty-four refractures through the bone or bones at the same location as the first fracture. Eighteen were in the forearm, four in the clavicle and two in the tibia. The age of these patients varied from seven to fourteen years in the forearm refractures, five to twelve years in the clavicular ones. The two boys who refractured their tibias were both seven years old. Of the forearm refracture cases, five were girls and thirteen were boys. The intervals between the first and second forearm fractures were as indicated in Table 1.



1A 1B

FIG. 1. A, fracture of lower third of radius and greenstick fracture of ulna in a ten year old girl; B, refracture eleven months later. Note both fractures are now complete.

For the clavicular fractures the intervals were five weeks, one year, one and a half years and three years. For the tibias the intervals were two and a half months and eight months. (Figs. 1 to 3.)

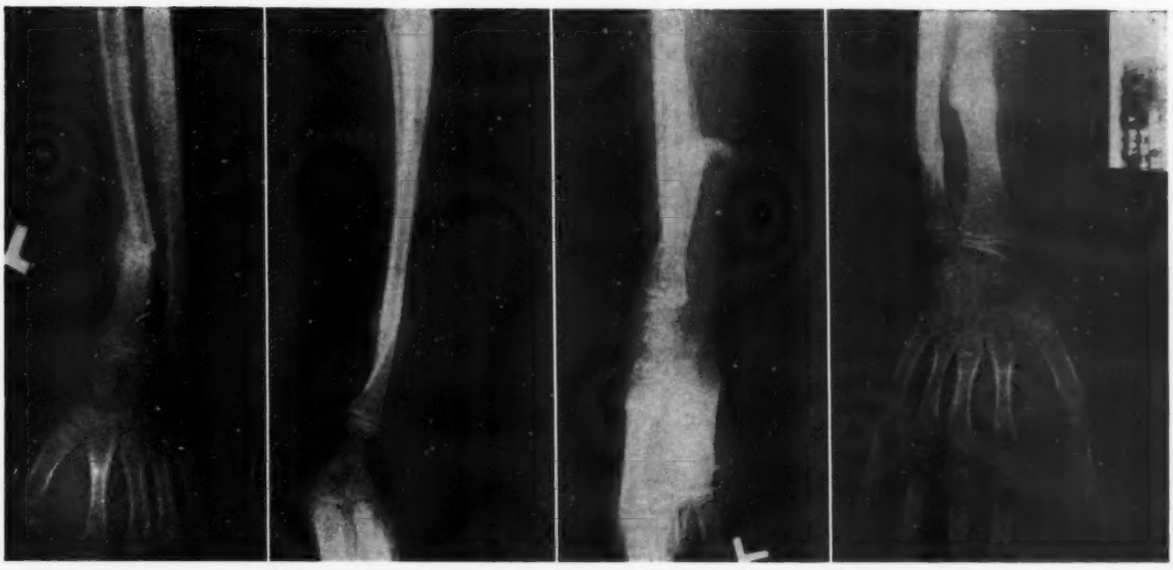
TABLE 1

| Age | Case No. |
|---|---|
| 4 wk. | 1 |
| 7–10 wk. | 4 |
| 11 wk.–3½ mo. | 5 |
| 5–12 mo. | 4 |
| 1½–5 yr. | 3 |

The patient, R. S., Case 7, who refractured his bone after four weeks, was a boy nine years old who suffered greenstick fractures of both bones in the middle third without displacement. No anesthetic was given and anteroposterior splints with sugar tong extensions around the elbow were applied. It was thought he had union in twenty-six days and splints were removed. On the twenty-ninth day the patient fell again and refractured his forearm and this time there was displacement of the fragments. His fractures were reduced and immobilized for eighty days. He managed to avoid another fracture although, of course, both times the period of immobilization was too short. Union can never be strong in such a short time. No one experienced in the care of these fractures would make this mistake.

One of the patients who refractured both bones of the forearm in the upper third at seven weeks had just had his cast removed and had his arm resting in a sling while awaiting a check-up x-ray. He had sudden pain and felt his bone crack. His doctor was called and after examination believed there was an increase in deformity. This is not obvious on the x-ray films but a crack through the callus of the ulna is apparent. The refracture was apparently produced by a rotary strain and the callus of the ulna was insufficient whereas the callus of the radius escaped by not resisting the force but by rotating with it. The damage here was not great but it indicates the caution necessary in handling these fractures. It also indicates the weakness of external callus designed for longitudinal strain against a rotary force.

Of the other cases refracturing forearm bones between seven and ten weeks Case 5 had received first fracture treatment elsewhere. Case 11 had fractures of both bones of the forearm in the distal third. The elbow was not immobilized and considerable volar angulation of the lower fragments remained. Nevertheless at six and a half weeks it felt clinically united and x-rays showed what was called solid union. At seven weeks the patient fell and refracture occurred. Six weeks' immobilization is not ordinarily too short for fractures in this



2A 2B

FIG. 2. Same case. Healing seven weeks and eight weeks after fracture (A) and refracture (B). Note in A that ends of radial fragments show no change in spite of abundant periosteal callus. Note callus distribution tends to straighten the bone. Crescentic shadow on lateral film (B) is an artefact.

location, but not immobilizing the elbow was a serious mistake as it continually subjects the ulna to rotary movement and delays healing. The angulation in this case in itself would determine the location of the refracture. The fourth case in this group was a doctor's son who was extremely active. His refracture of the middle third of the forearm occurred ten weeks after the original injury. Subsequently he is said to have suffered a third crack in the callus while wrestling with his brother. The small photographs of the x-rays do not reveal when this occurred but it is interesting to note that even though the radius was angulated the fracture line was obliterated three months after the second fracture. The ulna fracture line is still very distinct at four months. We think this is largely due to the rotary strain to which it is subject.

Of the groups that suffered refractures between eleven weeks and three and a half months, Case 8, P. R. (eight years old), is interesting because the patient was immobilized only twenty-five days for fractures of both bones of the right forearm only 1½ inches above the wrist. At three and a half months he fell and refractured his bones. One must speculate whether the union would have been stronger if the early mobilization were better.

Case 10 (age seven) in this group refractured both bones in the middle third at three and a

FIG. 3. Same case. Four years after first injury. Forearms compared in anteroposterior view. The injured radius is straight. The right has the normal curve.

half months. She had been immobilized forty-seven days and callus between fragments was visible at twenty-nine days. Once she had fractured the opposite radius and was thought to have some degree of osteogenesis imperfecta as she had blue sclerae and the cortex of the bone was relatively thin.

Case 14 (W. Y., age twelve) refractured both bones at three months. He had been adequately splinted for fifty-four days and the refracture is believed due to fairly severe trauma to an area which had not recovered normal strength. It emphasizes that forearm fractures in the middle third require protection for a long time in active boys.

Case 15 (M. K., four years old) suffered greenstick fractures of both bones in the middle third. Slight angulation of the radius persisted. At nine weeks his forearm was mobilized and eighteen days later he refractured his bones again, emphasizing the necessity for protecting healed fractures in children and the long time required for a fracture to heal to normal strength.

Case 16 (P. B., a girl, age eleven) fell, fracturing both bones in the distal third. There was some posterior angulation of the distal fragments which persisted after reduction. Casts were removed at six weeks. At this time the ulna fracture was indistinct but the radial fracture while showing callus was still indistinct. At ten weeks there was some deformity but full motion. At eleven weeks the patient fell and refractured through the old sites. Deformity and misinterpretation of the strength of the callus were factors subject to the surgeon's control in this refracture.

## COMMENT

The points of interest in this study concern the incidence, time and nature of bone healing, and the mistakes that have probably been factors in the refracturing.

The incidence of refractures of the forearm in children which we have found and that quoted by Buttner has surprised us by being as high as it is. The reason for this is undoubtedly the vigor with which children play and the lack of caution exercised because non-weight-bearing bones are involved.

Of great interest to us has been the emphasis that it has placed on certain aspects of bone healing. Review of this material sustains Bick's ideas on the distribution of callus in fractures of the long bones. The volume of the callus has been in proportion to the degree of displacement and secondly "its distribution has conformed to that arrangement which would most economically transmit the stresses and strains in the long axis." In children this mass or these masses of callus form quickly but its maturation seems slow so that normal strength is regained late, exactly how late we cannot say. The evidence from this study would seem to indicate that return to normal strength even in the bones of children requires more than a year.

The facts gained from such a small group as this does not permit one to do more than speculate and we must continue to consider what some of the factors in refracture are. Certainly it seems that angulation of the fragments favors it. Is the fact that one bone normally rotates on the other related to refracture? Our impression is that any rotation permitted by the method of immobilization favors delayed union of the ulna and consequently fracture through the callus. This is likely to occur when one fails to immobilize the elbow for lower third forearm fractures or when one attempts to immobilize the elbow with sugar tong splints or splints of inadequate length.

The study also brings out the need for long immobilization and protection of forearm fractures in children. It is also worthy of note that, as Buttner says, the callus which forms in children's fractures unlike that of adults does not remain tender until the union is quite strong. Consequently a clinical test which is useful in adults cannot be applied to children.

Another significant observation made by Buttner and confirmed in this series is that while the original fractures in one or both bones may be of the greenstick type, the refractures are always complete indicating that the healed bone may have lost some of its elastic quality.

## SUMMARY AND CONCLUSIONS

1. Refracture of bones occurs most often in the forearm in children. The incidence in the few reported series varies between 6 and 20 per cent.

2. Forearm refractures at the same location as the original fractures are most common

within three and a half months of the first injury but may occur as late as five years.

3. It is believed that a common error predisposing to refracture is overestimating the strength and value of periosteal callus. This produces rigidity before endosteal union occurs.

4. Union with angulation also predisposes to refracture.

5. The surgeon should protect forearm fractures in children well beyond the time required to achieve clinical and x-ray evidence of union.

REFERENCES

BUTTNER, A. Refractures in children. *Chirurg.*, 19: 347, 1949.

BICK, E. M. Structural patterns of callus in fractures of the long bones. 1. With reference to healing after internal fixation. *J. Bone & Joint Surg.*, 30-A: 141, 1948.

# FRACTURES IN METASTATIC CARCINOMA*

WILLIAM T. FITTS, JR. M.D., BROOKE ROBERTS, M.D. AND ISIDORE S. RAVDIN, M.D.
*Philadelphia, Pennsylvania*

ALTHOUGH cancer is widely considered one of our most important medical problems, relatively little attention has been given to fractures caused by cancer. The incidence of cancer is increasing in our population and those interested in the surgery of trauma should anticipate treating an increasing number of fractures due to cancer. It is our impression that the current aggressive attitude in the treatment of most forms of cancer has not been adopted for fractures due to metastatic cancer. All too often an attitude of hopelessness prevails and the patient is allowed to become bedfast for his remaining days, with severe pain and with rapidly diminishing morale. Although the prognosis is admittedly poor, vigorous treatment of the fracture will in most instances relieve pain, diminish the length of hospitalization, make nursing care easier and allow the patient to be moved for supplemental forms of therapy, e.g., irradiation. In a significant number of instances bone union does occur. We have found that intramedullary nailing is almost "tailor-made" for the treatment of many of these fractures, especially those of the femoral shaft. The following three cases illustrate what can be accomplished for these patients:

## CASE REPORTS

CASE I. M. S. (No. 65689), a forty-six year old man, had been treated at the Hospital of the University of Pennsylvania for hypernephroma of the right kidney, found in 1944. The tumor had metastasized to several bones. On November 28, 1949, he fractured the shaft of the right femur through an area of metastasis. An open reduction was performed the day after injury and a Küntscher nail inserted into the medullary cavity. Figure 1, a roentgenogram taken two months after injury, shows abundant callus at the fracture site. By six months the fracture had united. (Fig. 2.) The patient was relieved of pain by the operation, was out of bed after the first week, bore weight after the second month and worked for several months before his death, which occurred almost a year after the fracture.

CASE II. N. P. (No. 89226), a forty-one year old woman, fractured the neck of her femur on December 30, 1950, through an area of metastatic cancer. Four months earlier a radical mastectomy had been performed for carcinoma of the breast. The fracture was reduced and fixed with a Smith-Petersen nail. (Fig. 3.) The cancer had spread far into the head of the femur and it was believed that the nail must be driven close to the articular surface to give sufficient fixation. Because the lateral femoral cortex was also involved by the tumor, a McLaughlin bar was attached to the nail to give added stability. A sterilizing dose of irradiation was given over the ovaries and further irradiation was given over the site of fracture. Testosterone was given intramuscularly. The neck of the femur collapsed and the nail was displaced into the acetabulum. (Fig. 4.) In spite of this the fracture appears to have healed, the patient is bearing weight without pain and at the present time, almost two years after fracture, has no complaints.

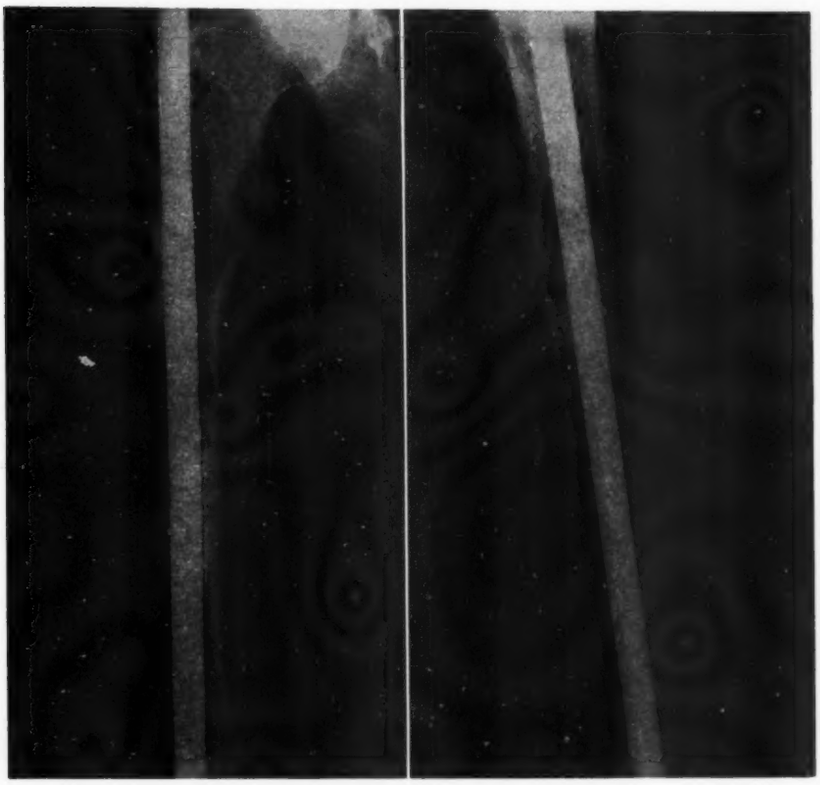
CASE III. C. S. (No. 79701), an eighty-four year old woman, fractured the shaft of her femur on October 20, 1948, through a metastasis from carcinoma of the bladder. Both she and her husband were willing to risk any treatment that, if successful, would permit her to return home and be out of bed. A Küntscher nail was inserted into the medullary cavity following open reduction (Fig. 5) and she was able to return home in two weeks, out of bed and able to bear weight.

The favorable results in these and other patients led us to analyze all fractures due to metastatic carcinoma seen in the Hospital of the University of Pennsylvania during the last ten years.

## ANALYSIS OF DATA ON FIFTY PATIENTS

During the ten-year period from July, 1942, through July, 1952, fifty patients were treated for fractures that had occurred in areas of metastatic carcinoma. These patients were treated on the General Surgical, Orthopedic and X-Ray services. Eleven of the patients were men and thirty-nine women.

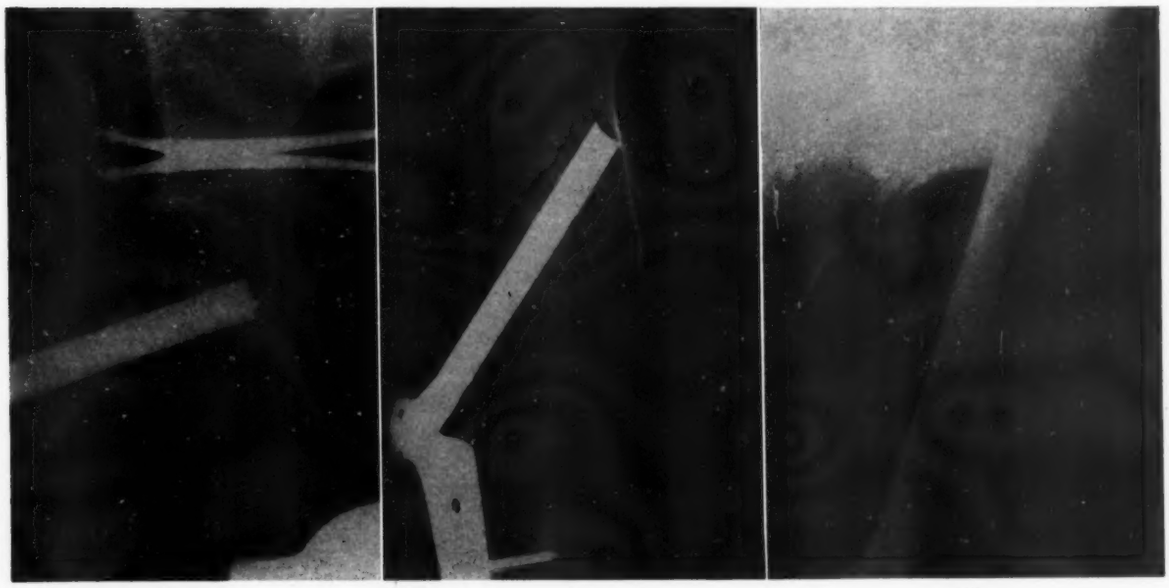
* From the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania, Philadelphia, Pa.



1 2

FIG. 1. Fracture of femur through metastasis from hypernephroma; roentgenogram taken two months after fracture.

FIG. 2. Same bone as in Figure 1; roentgenogram taken six months after fracture.



3 4A 4B

FIG. 3. Fracture of neck of femur through an area of metastasis from carcinoma of the breast; fixation with a Smith-Petersen nail. Roentgenogram taken immediately after insertion of the nail and showing involvement of the head and neck with cancer.

FIG. 4. Same bone as Figure 3; roentgenograms taken one year after fracture. Collapse of neck with displacement of nail into acetabulum. A, anterior view; B, lateral view.

FIG. 5. Fracture of femoral shaft through metastasis from carcinoma of the bladder; roentgenogram taken immediately after open reduction and fixation with an intramedullary nail.

*Location of the Primary Tumor.* The source of metastasis, as shown in Table 1, was carcinoma of the breast in more than half of the fifty patients. Carcinoma of the breast frequently metastasizes to bone and frequently causes fractures. This is apparent if we compare

TABLE 1
PRIMARY TUMOR IN FIFTY PATIENTS WITH FRACTURES DUE TO METASTATIC CARCINOMA

| Organ | No. of Cases | Per cent |
|---|---|---|
| Breast | 26 | 52 |
| Kidney | 7 | 14 |
| Colon and rectum | 4 | 8 |
| Lung | 3 | 6 |
| Thyroid | 2 | 4 |
| Prostate | 1 | 2 |
| Bladder | 1 | 2 |
| Parotid | 1 | 2 |
| Skin (malignant melanoma) | 1 | 2 |
| Pancreas | 1 | 2 |
| Testicle | 1 | 2 |
| Unknown | 2 | 4 |

two groups of patients with bony metastases, one with and one without fractures. (Fig. 6.) The data are compiled from cases reported in the literature[1–7] in addition to our own. Carcinoma of the breast was the primary tumor in 59 per cent of the fracture patients and in 47

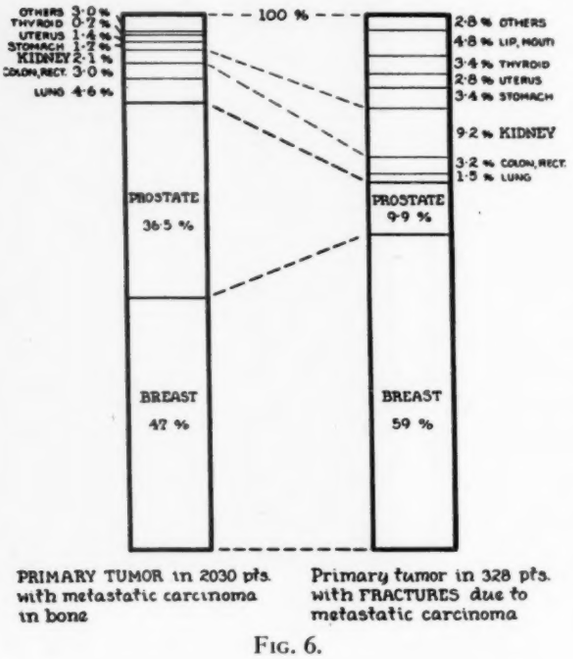



FIG. 6.

per cent of the non-fracture patients. Although carcinoma of the prostate also frequently spreads to bone, it seldom causes fracture. In our opinion the explanation lies in two facts: (1) metastases from carcinoma of the prostate are usually osteoblastic rather than osteolytic, and (2) prostatic metastases frequently involve the pelvis where pathologic fractures are uncommon. Although carcinomas of the kidney constitute a small percentage of tumors spreading to bone, when such lesions do metastasize to bone they are peculiarly liable to fracture and account for 10 per cent of the fractures collected. It is likewise noteworthy that bone metastases are more common from both the stomach and colon than they are from the thyroid, generally considered a common source of bone metastases.

*Location of the Fracture.* Bones of the spine and the femur are by far the most frequently fractured. (Fig. 7.) Table II shows the bones involved in the fifty-six fractures in our fifty patients. Data collected from the literature[1,3,5,6] and added to our own are presented in Figure 8, which compares the location of fracture in

two groups of patients with bony metastases: those with fractures and those without fracture. Bones of the spine make up a large percentage of each group. Although the femur is not commonly the site of metastatic carcinoma, when once involved it is frequently fractured. The pelvis is a common site of metastases but is rarely fractured.

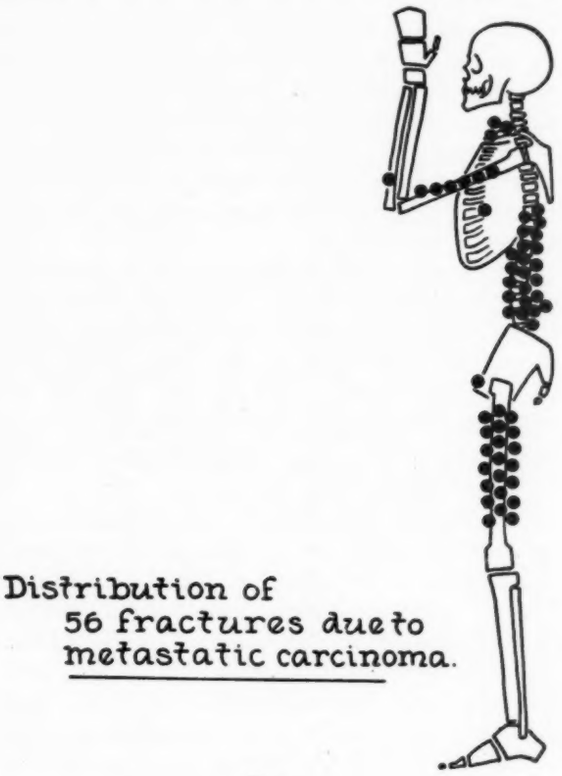



FIG. 7.

*Treatment.* We have done everything possible to avoid prolonged recumbency. In fractures of the femur the use of an intramedullary nail enables the patient to become ambulatory, reduces pain and gives an excellent chance for healing of the fracture. Fractures of the spine are a common problem and cannot be treated as definitively. We are loath to put patients with pathologic compression fractures of the vertebrae in plaster hyperextension jackets. They seem to do poorly and are uncomfortable. The majority of fractures of the spine do not require immobilization if they are discovered before compression has progressed very far. Pain ordinarily is encountered early in vertebral metastases. Local use of x-ray therapy is often of great benefit both in retarding the growth of the tumor and in giving symptomatic relief. In addition, endocrine therapy in suitable cases may also give dramatic improvement.

TABLE II
LOCATION OF FIFTY-SIX FRACTURES DUE TO METASTATIC CARCINOMA (FIFTY PATIENTS)

| Bone | No. of Cases | Per cent |
|---|---|---|
| Spine | 23 | 41.0 |
| Femur | 21 | 37.5 |
| Humerus | 6 | 10.7 |
| Clavicle | 3 | 5.4 |
| Rib | 1 | 1.8 |
| Pelvis | 1 | 1.8 |
| Ulna | 1 | 1.8 |

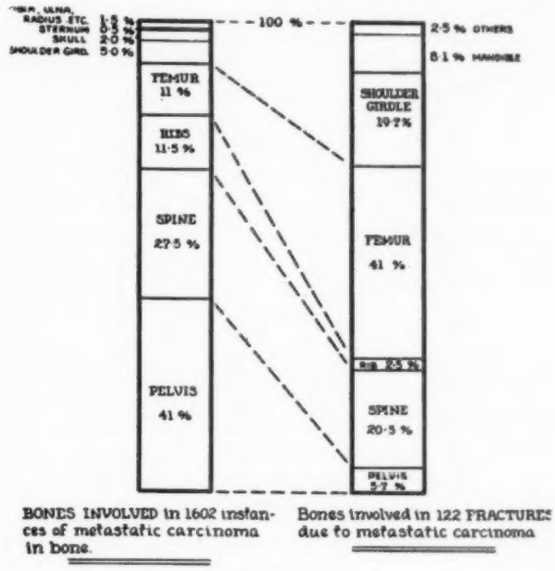

100 %
2·5 % OTHERS
8·1 % MANDIBLE
TEMUR 11 %
RIBS 11·5 %
SPINE 27·5 %
PELVIS 41 %
SHOULDER GIRDLE 19·2 %
FEMUR 41 %
SPINE 20·5 %
BONES INVOLVED in 1602 instances of metastatic carcinoma in bone.
Bones involved in 122 FRACTURES due to metastatic carcinoma

FIG. 8.

Irradiation has a large role in the treatment of pathologic fractures of almost all types arising from metastatic carcinoma. In general we use it locally over the bony lesion both for its relief of pain and its direct effect on the lesion. The majority of carcinomas that spread to bone respond to irradiation, although tumors arising in the gastrointestinal tract are a notable exception. In addition to local therapy, sterilizing doses of x-ray are often used in women who have tumors that may be benefitted by castration.

Endocrine therapy under certain circumstances may be as dramatic in its effect as x-ray. Therapy may consist of removing the source of normal sex hormones by castration or in supplying hormones of the opposite sex. Tumors of the breast and genital tract in both male and

female are particularly apt to be retarded by changes in the normal balance of sex hormones. It is now well known that prostatic carcinomas may be greatly improved by castration and estrogens, and that cancers of the breast at least in the period of active

TABLE III
DURATION OF LIFE AFTER FRACTURE

| | No. of Cases | Per cent |
|---|---|---|
| Less than 6 mo. | 24 | 48 |
| 6 mo. to 12 mo. | 12 | 24 |
| 12 mo. to 18 mo. | 5 | 10 |
| 18 mo. to 24 mo. | 2 | 4 |
| Over 24 mo. | 7 | 14 |

gonadal function, may be improved by castration and androgens whereas in later life estrogens may be beneficial. Huggins has presented evidence that adrenalectomy may give further benefit after the good effects of the usual hormone therapy have worn off. Total bilateral adrenalectomy was performed in two patients of this series with great symptomatic relief, one with carcinoma of the breast and one with carcinoma of the prostate.

Radical surgery in the form of amputation seldom is indicated for metastatic disease, but we did carry out amputation in one patient in whom a carcinoma of the colon spread to the ulna and resulted in a fracture. This bone did not unite and the lesion became progressively more painful and the arm less useful. X-ray had no effect upon it and eventually amputation was performed to relieve pain.

*Results.* All fifty patients have been followed up until their death or until August, 1952. Twenty-two per cent of the fractures healed and many more were healing at the time of the patient's death from progression of the disease. Table III gives the length of life after fracture for the fifty patients. Over half of the patients lived for more than six months and 14 per cent lived for more than two years. The fractures in all but two of the patients surviving two years or longer united solidly.

## COMMENTS

Carcinoma that has metastasized to bone has a poor prognosis, but this is hardly an indication for apathy on the part of the attending physician because many of these patients can be helped immensely at a most difficult period of their lives. We believe that open reduction and internal fixation is often indicated for such fractures of the long bones, not only because it may give the fracture its best opportunity to heal but also because of its effectiveness in relieving pain. As knowledge of tumors and their therapy progresses, further ancillary methods of treatment will undoubtedly be developed which will improve the results in these fractures. At present, hormones and roentgen ray therapy are the two main adjuncts to the treatment of the fracture itself and are especially effective in bony metastases from cancer of the breast.

The presence of an intramedullary nail does not preclude adequate irradiation therapy. Peltier and Nice[8] performed experiments which showed that the areas of the bone shielded by the nail lost only about 30 per cent of the effective irradiation. This can easily be compensated for by giving a larger dose of irradiation or using multiple ports.

It cannot be denied that intramedullary nailing spreads tumor cells in the medullary canal. It has been shown that red blood cells tagged with $P^{32}$ can be widely spread both distally and proximally in the marrow cavity of the tibia by the insertion of an intramedullary nail.[9] In none of our patients, however, have we noted any evidence of secondary growths along the medullary cavity and we believe that the advantages accruing from intramedullary nailing for many of these fractures far outweigh the risk of further spread of the tumor in a patient who already has metastatic disease.

## SUMMARY AND CONCLUSIONS

A review of the literature on fractures due to metastatic carcinoma and an analysis of fifty patients with such fractures treated at the Hospital of the University of Pennsylvania during the last ten years leads us to conclude that:

1. A hopeless "do-nothing" attitude is unjustified. Vigorous treatment of the fracture relieves pain, cuts down hospitalization, makes nursing care easier, and permits early ambulation.

2. Carcinoma of the breast accounted for the largest percentage of these fractures. Carcinoma of the prostate frequently spreads to bone but does not usually cause fractures.

Carcinoma of the kidney less commonly involves bone; but when it does, it is likely to result in fracture.

3. The spine, femur and bones of the shoulder girdle are most frequently fractured. Fractures distal to the knee and elbow are uncommon.

4. Open reduction and fixation with an intramedullary nail is often the treatment of choice, especially in fractures of the femoral shaft. Fractures of the spine due to metastatic carcinoma are usually best handled without plaster jackets or braces. Irradiation and endocrine therapy are sufficient to give the patient comfort and to limit the local progression of the tumor.

5. Twenty-two per cent of the fractures in our series healed although almost half of the patients died from progression of the disease within six months of fracture.

*Acknowledgment:* The authors wish to thank Dr. Paul Colonna and Dr. Roy Peck for allowing us to include patients treated by them in our series. Dr. Roy Greening and Dr. Richard Chamberlain were most helpful in selecting roentgenograms and in discussing the problems of irradiation in these patients.

REFERENCES

1. Welch, C. E. Pathologic fractures due to malignant disease. *Surg., Gynec. & Obst.*, 62: 735, 1936.
2. Ghormley, R. K., Sutherland, C. G. and Pollock, G. A. Pathologic fractures. *J. A. M. A.*, 109: 2111, 1937.
3. Geschickter, C. F. and Maseritz, I. H. Skeletal metastasis in cancer. *J. Bone & Joint Surg.*, 21: 314, 1939.
4. Turner, J. W. and Jaffe, H. L. Metastatic neoplasms. *Am. J. Roentgenol.*, 43: 479, 1940.
5. Smedal, M. I. and Salzman, F. A. The treatment of metastatic bone tumors. *Wisconsin M. J.*, 47: 675, 1948.
6. Golding, F. C. Metastatic tumors of bone. Diagnostic aspects. *J. Fac. Radiologists*, 1–2: 246, 1949.
7. Abrams, H. L. Skeletal metastases in carcinoma. *Radiology*, 55: 534, 1950.
8. Peltier, L. F. and Nice, C. M., Jr. Irradiation of bone lesions in the presence of metallic intramedullary fixation. *Radiology*, 56: 248, 1951.
9. Peltier, L. F. Theoretical hazards in the treatment of pathologic fractures by the Küntscher intramedullary nail. *Surgery*, 29: 466, 1951.

DISCUSSION OF PAPERS BY DRS. CASSEBAUM AND HAMILTON, AND FITTS, ROBERTS AND RAVDIN

Harold M. Childress (Jamestown, N. Y.): I greatly enjoyed this most interesting and valuable paper on surgical treatment of metastatic bone carcinoma. By such surgery hopeless individuals, many with severe persistent pain, may be given a tremendous lift both mentally and physically. I would like to describe a similar case recently seen.

The patient was a sixty-five year old white woman who in November, 1951, was hospitalized for pain at the left hip. Roentgenograms revealed a pathologic fracture at the proximal left femur. Traction was applied by her attending physician and she was given hormone injections but continued to experience severe localized discomfort.

She was first seen by me three months after hospital admission. Despite the fact that her left breast had been removed twenty years previously for carcinoma of that organ, the bone lesion from its x-ray appearance could well have been a localized fibrocystic condition. Biopsy, however, proved the pathologic condition of the bone to be that of a metastatic scirrhous-type carcinoma of the breast.

The roentgenograms were reviewed by two consultants who advised deep x-ray therapy and who were not in favor of any form of surgery. However, Dr. Bradley Coley of the Memorial Hospital for Cancer, agreed with me that surgery would probably be of great benefit to her.

Consequently in March, 1952, the entire proximal femur, including the head, was excised and an Eicher prosthesis inserted. One complication developed in that the stem of the prosthesis was too large for the intramedullary canal of the femoral shaft. Luckily this misfit was discovered before the appliance was driven too securely for removal. The stem was shortened 1½ inches by hacksaw and the final fitting was quite satisfactory.

In two weeks she was ambulatory and was discharged from the hospital three weeks later. She now lives alone in her home and walks with a cane. She has very little discomfort and her general condition has greatly improved. The ultimate outcome is not known at present, but this patient is quite happy and is most grateful.

James J. Callahan (Chicago, Ill.): I would like to inject the thought here that in metastatic lesions we have been using the intramedullary nail. Our experience is practically the same; the patients live about a year. Recently in solitary metastasis, like thyroid malignancy, we have been resecting the tumor and then inserting the intramedullary nail utilizing a bone graft to compensate for any deficiency in approximation. Our results have been far superior performing this procedure than by inserting the nail through the tumor with the danger of spreading the tumor cells.

Preston A. Wade (New York, N. Y.): I am very interested in Dr. Cassebaum's paper because in the last few years we have been plagued at New York Hospital with some recurrent fractures of both bones of the forearm in children. Dr. Harry

Kramer of New York Hospital has looked up these patients and has found nine well. Six of our own cases recurred, and three patients treated elsewhere were treated by us for the second fracture.

We found that these patients were from the ages of three to thirteen. Three had refracture after the plaster was removed in eight weeks, one a week, another a month and another four or five weeks. The other three occurred after sixteen weeks from the original fractures and after eight weeks from removal of the plaster. We have been in the habit of removing our plaster after an average of eight weeks.

The cases that were three and a half, four, and four and a quarter months were obviously not only the result of removal of the plaster. We have thought, however, that as a result of this we should keep our fractures of both bones of the forearm in children immobile for at least twelve weeks. I told this to Dr. Speed and he said, "The world doesn't know that, but I have known it for many years. But if we all knew that, there would be no purpose in these meetings, and that is why we are reporting our errors."

Of the three patients from other hospitals whom we treated, one was a child of two. The plaster had been removed at the end of three weeks and the fracture recurred two days later. One was a child of seven that had a plaster immobilizing elbow and wrist for some six weeks. The plaster was then cut down, freeing the elbow, and the second fracture occurred in the short plaster. We do not believe that the shorter plaster should be used, although I know some textbooks still say that after six weeks or even less the elbow may be released. As Watson-Jones says, this may cause more torsion on the fractured area.

I would like to commend Dr. Fitts on his presentation and say that we at City Hospital have also found satisfactory results in intramedullary nailing, particularly in femurs in metastatic carcinoma. I was surprised to hear him say that in 22 per cent he obtained bony union. I do not think we got as high a percentage as that. Even if bony union does not occur, these patients are much happier and much easier to take care of.

Thomas Bartlett Quigley (Boston, Mass.): One comment on Dr. Fitts' paper. We have been doing the same thing at the Peter Bent Brigham Hospital and we agree with everything he said. I would like to ask him one question. In some of these patients with metastatic carcinoma of the long bones, the carcinoma is sensitive to radiation and these patients have pain. When employing radiation to control pain, have you observed a variation in the natural history of the healing of the fracture in these cases?

George William Bancroft (Colorado Springs, Colo.): I would like to report a case of eight metastatic carcinoma fractures in one woman. Four and a half years after radical mastectomy she sustained a fracture of her right femur which was treated by traction and later with cast, and healed. During the next three years, she fractured both humeri, both clavicles, the other femur and refractured the first femur. These were all treated by traction and splints, and x-ray examination showed that they were all united.

The first femur had radiotherapy through a hole in the cast after it had been set. During the interim the patient did have x-ray therapy of her spine because one spinal body had collapsed, and she also had osteolytic evidence in her skull treated with x-ray, but actual treatment of the later fractures was nothing but immobilization, fixation and traction.

F. Walter Carruthers (Little Rock, Ark.): These papers indicate two things that I think should be emphasized. One of them is, we should make it on a comparative basis that refractures occur because of lack of proper time for complete immobilization for the fracture to reunite. Then on the other hand, we should sound a warning that other complications occur as the result of too prolonged immobilization producing what is commonly called Sudeck's syndrome or demineralization and decalcification. This latter condition is very apt to occur in prolonged immobilization of the forearm and hand and prolonged immobilization of the foot and ankle.

One of the first presentations here mentioned a separation at the head of the radius, in my opinion being released far too soon and obviously the deformity resulted. When refractures occur, if my memory serves me correctly, they do not recur in the sense I am thinking about, namely, at the site of the old fracture, except those that have not been given an opportunity to unite in the first place. However, if union has occurred and there is so-called refracture, it does not occur at the site of the old fracture. They either occur above it or below it. When a patient asks, "Am I apt to break my leg or arm in this place again?" I am always happy to reply, "No. If a fracture should occur, it will occur either above it or below it." That has been my experience in over thirty-five years of practice.

So the question arises which should be emphasized here, of a sufficient amount of immobilization and a sufficient amount of time to permit the fracture to unite. Obviously one cannot prevent fractures occurring if there are conditions in an individual that would produce a fracture. I therefore think we should not be misled in our deductions with regard to refractures but rather should bear in mind that the reason why there is refracture is because it was never permitted to reunite in the first place. Obviously I have had fractures occur at the site of the old fracture but it has always been

those that have not had an opportunity originally to unite solidly.

CHARLES HUGH MAGUIRE (Louisville, Ky.): I was particularly interested in the latter paper on metastatic carcinoma. I think it is best to decide what we are going to do with these metastatic lesions for two reasons:

First, I certainly agree with the original statement that cancer is on the increase. Secondly, as one who sees a good many cancer patients, we are seeing more and more bone lesions for one big reason: We are curing the primary lesions, and the patients are living long enough to develop bizarre skeletal lesions. We therefore had better decide how we are going to treat them.

HARRISON LLOYD MCLAUGHLIN (New York, N. Y.): The essayists are to be commended for their stand on the subject of intramedullary fixation for metastatic fractures. It is a good and humane procedure. It may be pointed out that it is also possible to insert radon seeds or removable radium elements into the center of the diseased area by way of the intramedullary device used for fixation. This may prove to have some advantages over external radiation therapy in selected radiosensitive lesions.

Dr. Cassebaum's paper has been discussed at length and there is complete agreement that the responsibility for all refractures in children occurring within three months of the original injury rests squarely upon the surgeon. Refractures occurring after six months are different. The point of healing constitutes the only hard and brittle segment in the normally resilient child's bone. It is only natural that another adequate injury will cause a fracture at this weak spot. This is confirmed by the level of later fractures in plated long bone fractures in children. That segment of the bone stabilized by the plate is relatively non-resilient. Later fractures occurring in the same bone are found consistently to occur at one or the other end of the plate. This phenomenon is sufficiently common to warrant elective removal of plates from the bones of children when the primary fracture is healed.

WILLIAM H. CASSEBAUM (closing): I would like to emphasize again that, as Dr. McLaughlin says, the doctor must share the blame when refracture occurs under three months. After that I am not so sure, and I would not advise seeking a remedy that may be worse than the condition. We have to take a calculated risk.

Dr. James Ewing once told a Scotchman that bad teeth, chewing tobacco and syphilis gave him a 20 per cent chance of getting cancer of the stomach. The Scotchman thought a minute and said, "Five to one; those are good odds; I guess I'll take them."

BROOKE ROBERTS (closing): I would like to answer two questions. One is on this problem of spreading the tumor by use of the intramedullary nail. Certainly on the theoretic basis it is possible but clinically these patients have had very widespread tumors at the time they had the fracture, and we have not seen what we believe to be a further spread due to use of the intramedullary nail. Of course, that is a clinical impression.

The other question is in regard to local treatment with x-ray, whether or not that has delayed healing of the fractures. It is always somewhat difficult to give a categorical answer to such questions. However, our radiotherapists have treated only for the relief of pain and have not used large tumoricidal doses. For that reason we have not thought that they had much effect on the healing of the bone itself.

# COMMON PITFALLS AND COMPLICATIONS IN HIP NAILING

F. P. MAIBAUER, M.D. AND W. H. HONOR, M.D.
*Wyandotte, Michigan*

IT is not the purpose of this paper to discuss the advantages of early open reduction of fractures of the hip, anesthesia or preoperative care, all of which have been treated thoroughly in previous papers presented before the American Association for the Surgery of Trauma, but (1) to discuss a simplified method for the accurate introduction of a nail into the neck of the femur and (2) to discuss the complications encountered in the treatment of open reduction of fractured hips.

In our hands, the use of a guide wire accurately placed and good x-ray technic has been sufficient to insert a nail properly in the neck of the femur.

As soon as the patient has been prepared for surgery, the first essential is an accurate reduction of the fracture obtained by a Leadbetter reduction if necessary. The injured leg is then placed in traction with the leg slightly abducted and the foot in internal rotation.

Now a point mid-way between the pubic tubercle and the anterior superior spine of the ilium is located. This point will be directly over the head of the femur. Then the greater trochanter of the femur is palpated and a Kirschner wire is laid on the anterior surface of the thigh between the mid-point and a point about 1 inch below the greater trochanter. Anteroposterior and lateral x-rays are taken, and while the films are being developed, this line is marked on the skin with a solution of silver nitrate, the wire is removed and the area prepared and draped for surgery.

In taking the anteroposterior x-ray it is important that the x-ray tube be directly over the wire so that the tube, wire and neck of the femur are in the same plane, and in order to do this a plumb line is used. If this is not done and the tube is at an angle, there will be considerable error on the x-ray plate.

If the reduction as shown on these preliminary x-rays is satisfactory, the operation is then carried out. A 6 to 8 inch incision is made on the lateral surface of the thigh extending downward from the greater trochanter and parallel to the shaft of the femur, the fascia and muscles split and the upper end of the femur exposed. A hole is drilled through the cortex of the femur and, using the line marked on the skin with silver nitrate as a guide, a guide wire held parallel to the top of the table is drilled into the neck and head and into the acetabulum in order to transfix the head. This is important especially in high intracapsular fractures; for when the head is first struck by the relatively blunt point of the nail, it may twist around and be displaced.

A second series of anteroposterior and lateral x-rays are now taken and if they show a proper placement of the guide wire, a Johannson modification of the Smith-Petersen nail of the proper length, which is determined by measuring the length of the wire projecting from the cortex and subtracting it from the known length of the wire, is driven over the guide wire. Care must be exercised in driving the nail over the wire so that the wire is not bent at the point of the nail, and subsequently driven ahead of the nail into the pelvis. After the nail is driven, anteroposterior and lateral x-rays are again taken; and if the position of the nail is satisfactory and if it is necessary, a 4- or 5-holed Thornton flange is attached to the nail and then securely screwed to the shaft of the femur. All screws must traverse both cortices of the femur.

As stated before, accurate reduction of the fracture, insertion of the nail and impaction are essential. In spite of all this, the following complications, in our experience, have occurred.

In two cases there was evidence that the material used was defective. The first case was a slipped femoral epiphysis in which a stainless steel wood screw was broken on insertion. The proximal fragment was removed and the case treated by fixation with two Kirschner wires.

The second was a fractured hip which was treated by the insertion of a Smith-Petersen nail and Thornton flange in which the machine screw used to attach the flange to the nail broke and had to be replaced by a new screw. These complications, at least in part, can be prevented by better control in manufacturing.

There was one case in which there was a

detachment of the machine screw which required replacement. A persistent draining sinus developed in one case which required removal of the nail and flange.

The Moriera screw was used in several cases but its use has been discontinued for the following reasons: (1) The large diameter of the screw and flanges causes too much destruction of bone substance allowing the screw to be extruded and (2) when the screw entered the head, it was rotated in spite of fixation with two fixation wires.

The last example is one in which the patient suffered a fracture of the right hip in 1949 which healed without complications. In 1951 the same patient suffered a fracture of the left hip which developed non-union and required an osteotomy in order to obtain bone union.

# SIMULTANEOUS BILATERAL TRAUMATIC DISLOCATION OF THE HIP

KELLOGG SPEED, M.D.
*Highland Park, Illinois*

ON May 25, 1946, a thirty-eight year old attorney riding in the rear seat was involved in a severe automobile collision. was lowered on to the covered floor and there a reduction was accomplished, accompanied by a feeling of slight crepitus. This was certified by

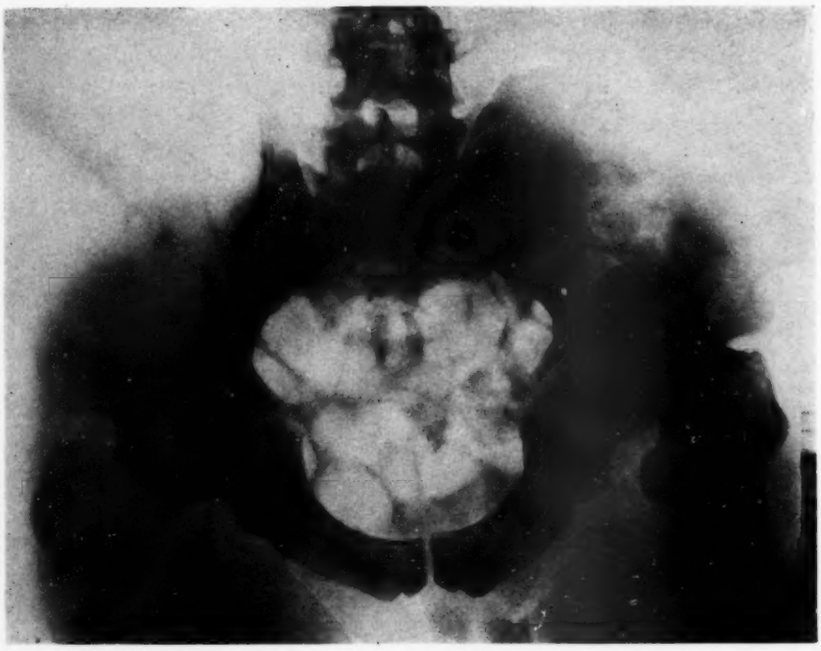


FIG. 1. Roentgenogram, May 25, 1946. Right posterior, left anterior simultaneous hip dislocation; fracture of the acetabular rim is seen on right side above the neck of the femur; none clearly shown in the left side, although present.

This patient was seen at St. Francis Hospital, Evanston, Illinois, three hours after injury. He presented multiple wounds and abrasions, a posterior dislocation of the left hip, with small fractures of the posterior superior acetabular edge, and an anterior dislocation of the right hip with less extensive acetabular rim fracture. (Fig. 1.) Catheterized urine showed no blood. He was appropriately treated for shock. When within two hours his systolic blood pressure rose to 120 mm., three unsuccessful attempts under general anesthetic, with the help of four assistants, were made to reduce the right hip by traction and manipulation. The head of the femur could be brought to the acetabular edge but not slipped into the joint. No curare was employed. For the fourth attempt the patient portable x-ray examination. (Fig. 2.) In spite of whole blood transfusion, falling blood pressure and shock were manifested, making it seem wise to postpone reduction efforts on the left hip at that moment. A plaster of paris hip spica was therefore applied, covering the right leg completely and the left leg (hip still dislocated) to the knee. This was intended to maintain the reduction of the right hip and to hold the left hip in *status quo* against untoward movements which might cause increased pain or add injury to the unreduced hip and surrounding tissues. Four days after reduction of the right hip the patient was removed to the Highland Park Hospital near his home in the adjacent county.

On June 5, 1946, the plaster encasement was

FIG. 2. Roentgenogram, June 3, 1946. Right hip reduced with evident fracture acetabular rim; left hip still in posterior dislocation.

removed. Under general anesthesia, with every care not to rotate or flex the right thigh, he was again placed on the covered floor. The posterior dislocation of the left hip was reduced by gentle but strong traction applied in the long axis of the leg by an assistant and myself, with counterpull and pressure on torso and pelvis, sustained by three other helpers, and followed by slow flexion and internal rotation of the left leg and thigh, accompanied by pushing-up pressure applied by one assistant on the greater trochanter of the femur. Reduction was confirmed immediately by portable x-ray, which examination also showed no interference with the previously reduced right hip. The patient was encased in a double body plaster of paris spica covering both legs, held in partial abduction and slightly flexed at the hip joints.

On July 15, 1946, this plaster dressing was removed. Pain in the left leg had practically subsided after reduction on that side. There was no foot drop on the left, but there was an area of diminished tactile and pain sensation along the distribution of the peroneal nerve, left. A roentgenogram on the following day confirmed fractures of the rims of both acetabula, with a few small fragments, slightly displaced and healing. It was hoped that the small amount of new bone formation might not untowardly influence future hip motions. No other fractures in pelvis or lumbar area were uncovered.

By August 1, 1946, the patient had good locomotion and only a slightly diminished tactile sensation on the outer side of the left foot. He returned to work September 15, 1946, after four months of relative quiet with slowly augmented weight-bearing and locomotion. Subsequent roentgenograms showed maintained reduction of both hips, no evidence of aseptic necrosis in the heads of the femora, but with some new bone formation becoming older and denser and retracting about both acetabula, especially the left. There was no restriction of active or passive motion in either hip, no pain after use or mild jars.

By June, 1948, he was able to play eighteen holes of golf on week-ends and volley ball on other days. On February 26, 1949, he complained of a small swelling in the right groin. There was found to be slight restriction of outward rotation of the right hip, but the roentgenogram failed to show any untoward change about the right hip joint except the previously noted small amount of new bone formation, now quite ancient, around the old minor fracture sites of the acetabular edge. He continued his sports and in February, 1950, sustained a partial rupture of the medial belly of the right gastrocnemius muscle, being

FIG. 3. Roentgenogram, May 15, 1952. Both hips remain reduced; no aseptic necrosis in the heads of the femurs; evidence of healed fractures of the acetabular rims; one projecting mass of new bone on right side not well seen at superior border of joint, but was shown on a lateral view not reproduced here.

compelled to use crutches for ten days. On October 4, 1951, a definite right femoral hernia was found. This was operated upon. A rather thick-walled sac medial to the femoral vessels was uncovered, with less than the usual amount of fat, no contents and a narrow neck opening into the abdominal cavity. Roentgenograms dated May 15, 1952, show normal hip joints (acetabula), no evidence of necrosis in the heads of the femora and still some new bone formation around the hip joints. (Fig. 3.) On the left side there had developed a projecting short horn-like area arising as an exostosis from the acetabular edge, as after a shelf operation. This does not interfere with a full range of bilateral hip motion.

Although simultaneous bilateral traumatic dislocation of both hips is as old as man himself, relatively few instances are recorded in written surgical records. In 1889 Kneer collected 1,068 instances of dislocations, 102 of which were of the hip. Only one was bilateral. In a forty-three year review of injuries cared for at the Sheffield Royal Infirmary, not a single instance of bilateral dislocation was found. At the orthopedic meeting in London, July, 1952, E. A. Nichol (Mansfield) reported 144 cases of hip dislocation, but made no mention of simultaneous bilateral instances. There are undoubtedly records of a large number of these double injuries buried in the memories and case histories of surgeons, but the number of reported cases is less than a hundred with not many over sixty detailed examples.

As we learn more of the blood supply of the hip joint, the upper end of the femur and especially its head, combined with roentgenologic information of aseptic necrosis of the bone after dislocation (and fracture), we now have a better and longer control of the real outcome. For that reason, this report has been withheld for six years to permit better documentation of this injury by means of follow-up study of any subsequent necrosis of the heads of the femora, the determination of the amount of new bone developing around the acetabular fractures and joint capsule and any evidence of so-called traumatic arthritis, or creeping substitution of dead bone. Time has also been allowed to determine the clinical result in range of joint motion, muscular power, action, nerve involvement and end results after the line of treatment adopted.

In the reported cases, males represent about 90 per cent and females 10 per cent. The mortality as related, especially higher in the ancient

cases, is about 4 per cent, depending upon the severity of the injuries and complications sustained. Of these complications, fractures of the acetabular rim, including the cartilaginous cuff, are the most frequent, the size of bone fragments and their displacement varying, but in recent times confirmed by roentgenologic examination. Sciatic or other nerve complications are quite frequent and often of minor degree. Their prognosis is good, depending on early recognition in diagnosis and accessory splinting employed during the waiting period, as for recovery from drop foot. Complete nerve severance naturally calls for operation, with a none too good prognosis if the nerve has been severed and sutured.

Necrosis of the head of the femur is infrequent among modern case reports. This bids us to pause in too literal an interpretation of clinical prognosis of necrosis of the head of the femur by possible interference with the anatomical blood supply of the femur via the ligamentum teres and other sources, torn or stretched capsule, hemarthrosis and compression from delayed reduction. New bone formation about the acetabular fragments of varying displacement or along the denuded acetabular edge or in the torn capsule of the joint, may not become causes of restricted joint motion and may, in fact, become partly absorbed or even disappear roentgenologically under treatment which spares the joint overuse or too active motion and weight-bearing. It is my opinion that the joint should be rested for six or eight weeks. Light active motions may be allowed, not coupled with weight-bearing. A diffuse calcification laid down in the capsular tissues may render them inelastic and non-pliable, limiting motion. These may be avoided by slowing progressing non-irritating use in prolonged convalescence.

The causes of simultaneous bilateral traumatic dislocation may be reduced to a few mechanisms: (1) A heavy blow in the lumbar or sacral region with legs in spread eagle or different positions. In this may be included crushes between massive surfaces, legs in varying positions. (2) Falls from a height with legs often spread apart but not exclusively so. (3) A combination of (1) and (2) plus twists of the legs or torso in a sudden cessation of movement, as may be experienced in an auto crash by individuals sitting in either front or rear seat. They may be suddenly precipitated forward by their inertia as the vehicle comes to a sudden stop, their legs in almost any position. Often the legs are spread apart or the twisting of the torso throws one into adduction, one into abduction, with or without rotation. Warning should be issued to automobile riders not only to refrain from sticking elbows and arms out of windows, but also to sit up straight, not to cross legs, and in case of collision to slide off the seat as a unit, if possible, using arms to protect face and avoiding abduction of thighs. A fractured rib or injured arm is less unprofitable than a dislocated hip or fractured patella or femur.

The pathogenesis need not be elaborated. Mostly, the dislocations are either posterior or anterior or a combination in position, depending on the mechanism of the applied violence, position of the legs and torso and any twisting or rotation of either. In the reported instances double posterior dislocations (which may be high iliac or lower ischiatic) represent 50 per cent. Double anterior dislocations (which may be high pubic or obturator) represent 10 per cent. The remaining 40 per cent are various combinations of the two principal types.

Kneer's case (1851) described a fifty year old male with bilateral posterior dislocations received while working in a sand pit, reduced eight days later. Randle's case (1921) was a male, forty-four years old, who was pushed into a pit by a truck, fell 22 feet, during which he turned a complete somersault, landing in a stooped position on both feet, legs slightly apart. His feet were rotated inward and the inner borders of his knees were in contact when he was rescued. One os calcis was fractured. Hill's case (1929) was a forty year old male who fell 40 feet off the roof of a house. He sustained fractures of the skull and spine, with dislocation of the left hip backward, the right hip forward, into the obturator foramen. Walter Stern's case (1921) was a twenty-seven year old female injured in an automobile wreck while riding as passenger in the front seat, which was movable and adjustable. As the automobile skidded, the driver was thrown into her lap. The whole seat was thrown out, clear of the car, carrying them as on a toboggan across the wet street, so that their feet struck against the curb on the soles and heels of their shoes. The woman had also injured her right foot, had a right peroneal palsy, with anesthesia in the toes and pains in the calf of the leg, which were

so severe that injury of the hips and spine was overlooked. Several physicians had seen her and after seven weeks one found a posterior dislocation of the right hip and took her to Dr. Stern. It was only at the start of reduction of this right hip that Dr. Stern noticed the left hip was similarly afflicted. A roentgenogram confirmed the bilateral dislocation, with only a very small chip fracture of the acetabular rim. Both hips were finally reduced, seven and one-half weeks after the accident. A splint was applied on the drop foot and the legs were bound together, pillow between. After one week circumduction of the hip (passive) was started. Stern believed that his case was the fortieth on record up to 1921.

Taylor's case (1938) was a fifty year old male injured while on the seat of a lorry in head-on collision. He sustained a right forward and a left posterior dislocation. After reduction, a Steinmann pin was used for leg traction for three weeks, followed by exercises and intermittent traction. Taylor noted that inguinal hernia, hydrocele and peroneal paralysis were sequelae, and that the average disability of reported instances of bilateral dislocation was eighty-one days. Cornacchia's patient (1940) was a thirty-five year old female who jumped out of a third story window. She had a sciatic dislocation on the right side and an iliac on the left, with complete paralysis of the sciatic nerve, from which she made recovery in sixteen months. Cornacchia found fifty-five cases in the literature. Magruder's patient, male aged twenty-six, fell forward off a moving handcar and was run over, violently flexing his thighs on the abdomen, causing internal rotation of legs and feet. His knees touched on their anterior surfaces. He had a double posterior dislocation, and there was great difficulty in reducing the left hip. He was out of bed in a few days although he also had a pneumonia.

Jaroschy demonstrated a bilateral case in a female, twenty years old, in Prague in 1921. This had been followed by much bone formation about the hip joint. Chace's report in 1912, was of a male, aged twenty-nine years, who was bending over, hands almost touching the ground, when a 1,000 pound weight fell 30 feet to strike him on the back while both hips were flexed, adducted and rotated inward. A double ischiatic dislocation was found, but Chace said nothing of acetabular rim fracture. James Lewis' patient (1904) was an eighteen year old male driver in a coal mine. He was thrown in front of a loaded coal car while riding on the bumper, was run over and sustained a bilateral posterior dislocation. This author gives a report of thirty-two cases, 1830 to 1904, and gives Six's Table (Wurtzberg Thesis, 1891).

The article by Paul Niehans is seventeen pages long. He collected seventeen cases, starting with Singowitz' of Danzig in 1830, which he described in full. This case is duly credited in my Textbook on Fractures and Dislocations. Ach's patient (1929) fell out of his automobile, had posterior dislocation of both hips with fracture of the left acetabular lip. Joint movements and massage were started eight days after reduction and there was good function in both hips five months later. One of the best reports is by Marquardt, who not only gave his own—a female of thirty-three with a right-sided pubic and left-sided iliac dislocation—but said that in 1936 there were fifty-three known bilateral dislocations. He described the mechanism, studied all the ancient cases and worked out some enlightening line drawings.

For treatment we suggest: X-ray for position and any fracture; if shock is absent, reduce at once by manipulation, using general anesthesia. There must be plenty of assistants to help, especially if the patient is lowered onto the floor. Weight-bearing is delayed especially in the face of minor accompanying fractures, but active motion of the hips may be allowed in from four to eight weeks after reduction. A careful follow-up roentgenologic study for at least two years is indicated, to determine any excess new bone formation or aseptic necrosis of the head of the femur. Such sequelae as inguinal or femoral hernia, or unyielding sciatic nerve lesions may require operation at a later period.

SUMMARY

1. Dreaded necrosis of the caput femoris has not followed this instance of simultaneous traumatic dislocation of both hips with minor fractures of the acetabular rim.

2. Fractures of the acetabular rims have healed with no apparent blocking of hip joint motions up to six years after dislocation. Later changes may appear, especially in a man so athletically inclined.

3. When shock exists, with bilateral dislocation of the hip, one side may be reduced at a time, without too great an interval (ten days).

4. Aseptic necrosis of the head of the femur does not follow every hip dislocation, even when unreduced ten days. The ratio of acetabular fracture, the interference with articular circulation from tension or malposition, hemarthrosis, capsular tear or pressure and ligamentum teres laceration are all factors contributing to possible necrosis of the head of the femur. Local pressure and thrombosis seem most important to me.

5. There is no doubt that fragments of accompanying acetabular fracture may lead to difficulties of obtaining and holding reduction, or lead to a tendency to recurrence or subsequent restriction of joint motion. I am in accord with Dr. Urist' excellent study of this problem and believe some instances require operative fixation of the acetabular lesion.

6. Putting the patient in a position where one may apply one's utmost physical power under control is the wisest course in reducing dislocation of the hip. Try the floor.

7. Undoubtedly, prolonged rest after reduction of hip dislocation with slowly increasing active motions of the hip and long delayed weight-bearing favor a good prognosis and a minimum of new bone formation which may restrict hip motion with or without acetabular rim fracture.

8. A young athletic male is a wonderful bit of mechanism.

## REFERENCES


Ach, C. Doppelseitige traumatische Hüftgelenksluxation. *München. med. Wchnschr.*, 77: 579, 1930.

Barclay, S. Bilateral dislocation of hip joint. *New Zealand M. J.*, 48: 310, 1949.

Chace, A. E. Traumatic dislocation of both hips. *New York State J. Med.*, 95: 171, 1912.

Claridge, J. D. Bilateral traumatic dislocation of the hips. *Proc. Inst. Med. Chicago*, 10: 325–326, 1935.

Cornacchia, M. Casistica rara; un caso di Lussacione traumatica bilaterale dell'anca. *Chir. d. org. di movimento*, 33: 32–36, 1949.

Dotter, W. E. and Hammond, G. Bilateral fracture-dislocation of hip joints; case report. *Guthrie Clin. Bull.*, 20: 5–11, 1950.

Eunike, K. W. Zwei Fälle von doppel-seitiger traumatischer Hüftluxation. *Deutsche med. Wchnschr.*, 44: 40–41, 1918.

Hill, H. G. and Penn, J. Bilateral traumatic dislocation of hips; case report. *South. M. J.*, 22: 972–974, 1929.

Hoffman, C. S. Dislocation of both hip joints. *West Virginia M. J.*, 3: 266–267, 1909.

Jaroschy. Einen Fall von doppelseitiger Luxation des Hüftgelenks.

Kneer, F. Ueber 32 traumatische Hüftgelenks-Luxationen. *Brüns Beitr. klin. Chir.*, 4: 513–536, 1889.

Lewis, J. H. Double traumatic iliac dislocation of the hip. *Ann. Surg.*, 40: 735–738, 1904.

Magnus, B. Ueber den Zustand der Gelenke nach reponierten Luxationen. *Arch. f. klin. Chir.*, 148: 55–56, 1927.

Magruder, E. M. Simultaneous dislocation of both hip joints in the same patient. *Virginia M. Semimonth.*, 21: 317–318, 1916.

Marquardt, W. Die doppelseitige traumatische Hüftluxation. *Arch. f. Ortbop. u. Unfall-Chir.*, 37: 189–201, 1936.

Niehans, P. Ueber traumatische Luxationen beider Hüftgelenke. *Deutsche Ztschr. f. Chir.*, 27: 467–494, 1888.

Randle, A. A case of double dorsal traumatic dislocation of the hip. *Lancet*, 1: 589, 1921.

Speed, K. A Text-Book of Fractures and Dislocations, 4th ed., p. 827. Philadelphia, 1942. Lea & Febiger.

Stern, W. G. A case of bilateral dislocation of the hip joint. *J. A. M. A.*, 76: 1496–1497, 1921.

Taylor, R. G. A case of bilateral traumatic dislocation of the hips. *Brit. J. Surg.*, 27: 605–607, 1940.

Urist, M. R. Fracture-dislocation of the hip joint. *J. Bone & Joint Surg.*, 30: 699–727, 1948.

Von Bardeleben, A. Geburt bei Hüftgelenksluxation. *Ztschr. f. Gebürtsh. u. Gynäk.*, 58: 519–522, 1907.

Wohlberg. Ein Fall von traumatischer doppelseitiger Luxatio perinealis. *Deutsche Ztschr. f. Chir.*, 72: 342–343, 1904.

Wood, J. R. Case of luxation of both hips. *New York M. Times*, 2: 33–34, 1852.

# TRAUMATIC DISLOCATION OF THE HIP

RALPH K. GHORMLEY, M.D. AND ROGER SULLIVAN, M.D.
Section of Orthopedic Surgery, Mayo Clinic Fellow in Orthopedic Surgery, Mayo Foundation
*Rochester, Minnesota*

TWO names are outstanding in the early English literature on traumatic dislocation of the hip. They are Sir Astley Cooper and Henry Jacob Bigelow. Each of these wrote at length on the anatomic aspects of this lesion, accurately describing methods of reduction and giving classifications of the lesions. However, neither of them, so far as we are able to determine, discussed the end results of their treatment of this condition. It is to the end results of this serious injury that a great deal of attention has been drawn in the past two decades.

It is hard to separate dislocations of the hip from fractures of the acetabulum, the latter being a complicating factor in many cases of dislocated hip. The exact incidence of fracture of the acetabulum and its significance with regard to prognosis are important. Several papers published during the past few years have served to emphasize the importance of careful study of end results. Such papers, no doubt, will stimulate all of us to greater initial efforts at gaining a perfect reduction in order that, if possible, a better end result may be obtained.

Four of the more recent articles available on traumatic dislocation of the hip are summarized in Table 1. Follow-up data were obtained in 128 of 193 cases by Speed and Stewart, in 116 of 204 cases by Thompson and Epstein, in 101 cases by Armstrong, and in sixty-six of seventy-six cases by Paus.

From a review of these articles, particularly the papers of Speed and Stewart and of Thompson and Epstein, it is obvious that early reduction of the dislocated hip is a most important factor in recovery with a good end result.

Speed and Stewart state, "The total figures of the cases reduced by manipulation within the first 12 hours reveal that 37 of 42 received an excellent or good result and 5 fair or poor. In the second 12 hours there were 11 excellent or good and 4 fair or poor. After 24 hours there were 33 cases all rated as fair or poor regardless of the type or grade of dislocation."

The late results in cases in which open reduction was carried out were less satisfactory. In 71.4 per cent of such cases in Speed and Stewart's series, traumatic arthritis developed. Thompson and Epstein point out the same fact, that early reduction of manipulated dislocations produces significantly better results.

TABLE 1
END RESULTS OF TRAUMATIC DISLOCATION OF THE HIP

| Author | Year | Cases* | | | | |
|---|---|---|---|---|---|---|
| | | Total | Traumatic Arthritis | | Aseptic Necrosis | Injury to Sciatic Nerve |
| | | | Number | Per cent | | |
| Speed and Stewart.... | 1952 | 128 | | | | |
| | | (90)† | 44 | 48.8 | 14 | 17 |
| | | (28)‡ | 20 | 71.4 | 11 | |
| Thompson and Epstein.... | 1951 | 116 | 37 | .... | 27 | 26 |
| Armstrong.... | 1948 | 101 | .. | 15 to 100§ | "Small number" | 7 |
| Paus..... | 1951 | 66 | .. | 28** | 1 | 0 |

* Cases in which follow-up reports were available
† Non-operative reduction
‡ Open reduction
§ Depending on severity of damage (varying from simple dislocation to central fracture-dislocation)
** Symptoms present

Armstrong's study of 101 dislocations was based on an admittedly short follow-up period—admittedly too short for any final results from the standpoint of traumatic arthritis or aseptic necrosis (two to four years). He states that "complete recovery, as judged by clinical and radiographic examination was observed in 76 per cent of simple dislocations, 63 per cent of dislocations with fractures of the acetabular rim, and 40 per cent of dislocations with fracture of the femoral head, in no case of dislocation with fracture of the acetabular floor was recovery complete." He further states, "Early traumatic arthritis developed in 26 per cent of patients—in 15 per cent of simple dislocations, 25 per cent of dislocations with fracture of the

acetabular margin, 60 per cent of dislocations with fracture of the femoral head and 100 per cent of dislocations with fracture of the acetabular floor."

Urist,[7] in an article read before this organization, reported on fifteen patients who had dislocation of the hip without major fractures of the head of the femur or acetabulum. Nine were followed only two years and of these, four had no symptoms and five had "mild pains." Urist,[8] in another article, reported on twenty-seven patients with fracture-dislocation treated by open operation or by conservative methods. After a two-year follow-up period he concluded:

"1. Fractures of the posterosuperior rim of the acetabulum with displacement should be repaired as carefully as any other fracture of a weight bearing joint.

"2. Fractures of the posterior rim of the acetabulum associated with dislocation of the hip, should be treated by cautious closed manipulation followed by open reduction and internal fixation of the fracture through a posterior approach.

"3. Dislocation of the hip joint associated with comminuted fracture of the acetabulum should not be subjected to closed manipulation. The only safe and efficient method of treatment is primary open reduction as soon as the patient is in fit condition for a major procedure. When the goal of the operation is replacement of large fragments and exploration of the sciatic nerve, the posterior approach is required; when replacement of the head of the femur and erosion of the joint surface in preparation for fusion are indicated, the anterior approach is advisable.

"4. Fractures of the head of the femur except in cases in which it is necessary to reduce the size of the head or to excise the intra-articular fragments, are best treated by conservative methods."

Our series consisted of seventy-three patients who had dislocation or dislocation with fracture and who were seen at the Mayo Clinic in the years 1923 through 1948. Twenty-seven of these patients were treated primarily at the clinic, while the remainder were seen at the clinic at varying intervals following their injuries. Obviously the latter patients represented a more complex group, as it is unlikely that we would have seen a patient whose dislocation had been successfully reduced and who was getting along well. These patients, therefore, presented a proportion of complicating injuries that is somewhat higher than in any of the series we have reviewed.

In our series of seventy-three patients there were twenty-four with sciatic palsy of varying degrees of severity, thirty-seven with fracture of the acetabulum, seven with fracture of the femoral head, two with fracture of the femoral neck, and two with evidence of myositis ossificans.

A review of the records and follow-up letters on these patients indicated that thirty-eight had some degree of traumatic arthritis and ten had definite evidence of aseptic necrosis. Not all patients with so-called traumatic arthritis could be said to have evidence of aseptic necrosis; it simply could not be stated definitely, from the evidence available, whether there was such a complication or not.

Follow-up information was obtained on seventeen of the patients treated primarily at the Mayo Clinic, and of these, sixteen were treated by closed reduction and one was treated by open reduction. The last-mentioned patient was a boy who had, as a complication, a slipping of the capital femoral epiphysis. The epiphysis was fixed with Knowles' pins, and a good result was obtained.

Eight of the seventeen patients had injury of the sciatic nerve. One of these patients recovered completely, four were improved and three were unimproved. Four of the seventeen had pain, eight had a limp, and nine had normal movement of the hip. One patient had myositis ossificans.

Various surgical procedures were carried out in the entire series of cases as follows: open reduction with or without nerve exploration, ten cases; cup arthroplasty, eight cases; arthrodesis of the hip, six cases; osteotomy and cheilotomy, two cases.

Among the patients who underwent late open reduction and on whom satisfactory follow-up data were obtainable, none had a good result except one patient on whom a shelf operation was done for recurrent dislocation. This patient had no pain and a minimal limp at the time of follow up. Two patients on whom cheilotomy was done had moderate pain but a severe limp, one of them using crutches or a cane.

Of the eight hips (seven patients) on which Vitallium-mold arthroplasties were done, four represented satisfactory results. Another patient underwent bilateral arthroplasty else-

where with a poor result, and two patients were not traced.

Of the six patients who were treated by arthrodesis of the hip, two had no pain and were satisfied, two had slight pain and there was a question whether the arthrodesis was solid, and two were not traced.

COMMENT

It seems clear from our own observations as well as from a careful study of the aforementioned articles that in simple dislocations without fracture, early reduction by manipulation, that is, within twelve or at most twenty-four hours, offers the best opportunity of obtaining a good end result. In patients coming to the surgeon with dislocations unreduced, open reduction must be done as a rule but a guarded prognosis must be given. In patients with fractures of the acetabulum closed reduction is probably the best method. Thompson and Epstein at least hint this, indicating that better results were obtained when posterior fractures of the hip were treated by closed methods; however, when fragments of bone were loose in the joint, better results were obtained by removal of the fragments.

Speed and Stewart do not emphasize the importance of open reduction and fixation of fragments although they report some good results after such treatment. Their comment, however, is as follows:

"It appears, therefore, that early reduction is the most essential feature of treatment. If operation is necessary and the general condition will not permit it within the first 12 to 24 hours, then reduce the fracture and maintain reduction by traction. Surgery may safely be delayed for a week or ten days. It becomes more apparent that treatment of the capsule and its inherent blood supply is of more importance in preserving the integrity of the hip and preventing complications than reduction of the fractured acetabulum."

From these and other observations as well as our own it seems that any estimate of true end results from open reduction and removal or replacement of fragments must await further experience, and trial of other methods may produce better results. Certainly the end results from manipulative reduction alone leave much to be desired, and any method to improve these results should be welcomed.

As for prevention of aseptic necrosis by prolonged prevention of weight bearing, there seems to be agreement among most of these writers that prolonged prevention of weight bearing does not necessarily prevent aseptic necrosis.

Myositis ossificans which is thought to be a common complication of dislocation of the hip is relatively infrequently seen. No one has offered a suggestion as to its prevention.

*Injury to the Sciatic Nerve.* This complication may be easily overlooked unless a careful examination of the extremity is carried out. One should test not only motor power in the muscles of the foot and knee but also sensation in order to determine the extent of the injury. If there is evidence of severe damage to the sciatic nerve, exploration of the nerve is frequently advisable to determine the extent of the injury and to try, if possible, to improve the function of the nerve; injuries of lesser degree may be watched for evidence of improvement. In our own experience improvement took place in half of the cases but complete recovery was noted in only one case, and there was no evidence of improvement in three cases. Speed and Stewart reported variable degrees of recovery in seventeen cases: in three there was no recovery; in three, complete recovery; in six, 50 per cent recovery; in one, 25 per cent recovery; in two, 75 per cent recovery; and in two the patients were not traced. They did not note any relation between a good recovery of hip function and a good recovery of nerve function.

Thompson and Epstein did not attempt to draw any conclusions as to the incidence of injury to the sciatic nerve among posterior dislocations. However, they found no instance of recovery among three patients who sustained severe injury to the sciatic nerve. Fourteen patients with injury to the peroneal portion of the nerve recovered in periods ranging from three months to two years. One patient experienced partial recovery after two years. Six patients who underwent open reduction had injury of the sciatic nerve but no impingement of the nerve was found in any of these instances. They did not attribute any improvement in the nerve to operative treatment.

It seems, therefore, from the evidence in these reports, that there is little to prove that exploration of the nerve is indicated frequently. However, we doubt that this report covers a

sufficient number of cases to prove or disprove the value of careful exploration of the sciatic nerve.

Again in these cases, if operation is to be performed, it should be performed early, as there is certainly nothing to be gained in waiting to relieve damage to a nerve if such relief can be accomplished by operative treatment.

## SUMMARY AND CONCLUSIONS

1. Early reduction by manipulation is indicated in all cases of traumatic dislocation of the hip.

2. In cases in which fracture is a complication, reduction should be accomplished as early as possible, with careful open operation to remove fragments of bone and to replace larger fragments of the acetabular margin.

3. Evidence of injury to the sciatic nerve should be looked for, and in some instances exploration of the nerve should be performed.

4. Aseptic necrosis is a late complication. Nothing known to date will prevent the occurrence of this complication at times.

5. Some element of traumatic arthritis develops in at least half the cases of uncomplicated traumatic dislocation and in most of the cases in which there is associated fracture.

6. Myositis ossificans is not a common complication.

## REFERENCES


1. Armstrong, J. R. Traumatic dislocation of the hip joint: review of one hundred and one dislocations. *J. Bone & Joint Surg.*, 30: 430–445, 1948.
2. Bigelow, H. J. 1. The Mechanism of Dislocations and Fracture of the Hip. 11. Litholapaxy; or, Rapid Lithotrity with Evacuation. Boston, 1900. Little, Brown & Company.
3. Cooper, A. A Treatise on Dislocations and Fractures of the Joints. Boston, 1844. Press of T. R. Marvin.
4. Paus, B. Traumatic dislocations of the hip: Late results in 76 cases. *Acta ortbop. Scandinav.*, 21: 99–112, 1951.
5. Speed, J. S. and Stewart, M. J. Dislocation and fracture dislocation of the hip due to trauma: end result study. Unpublished data.
6. Thompson, V. P. and Epstein, H. C. Traumatic dislocations of the hip: A survey of two hundred and four cases covering a period of twenty-one years. *J. Bone & Joint Surg.*, 33: 746–777, 1951.
7. Urist, M. R. Injuries to the hip joint: Traumatic dislocations incurred chiefly in jeep accidents in World War 11. *Am. J. Surg.*, 74: 586–595, 1947.
8. Urist, M. R. Fracture-dislocation of hip joint: Nature of traumatic lesion, treatment, late complications, and end results. *J. Bone & Joint Surg.*, 30: 699–727, 1948.


## DISCUSSION OF PAPERS BY MAIBAUER AND HONOR, SPEED, AND GHORMLEY AND SULLIVAN

George James Curry (Flint, Mich.): It has been a privilege to have had the opportunity to listen to three such excellent presentations. I would like to direct my remarks to the paper of Dr. Maibauer, having to do with simplicity, as I presume his objective is meant to be, relative to the pinning of hips; for that I wish to compliment him.

The use of a guide pin in our group has been a continuous procedure over these past fifteen years. There have been some 900 or more hips operated upon during that period of time, and I do not believe that I can recall a single femoral neck fracture that had an arthrotomy.

I would like to correct, perhaps, the impression that Dr. Maibauer may have given when he stated that this was an open reduction. It is an open reduction for an intertrochanteric fracture, that I presume he meant to imply, and a closed reduction of the intracapsular fracture, pinned through the lateral approach.

I think all of us in this group who have had to do with the pinning of hips have had many things happen to us. The guide pin is a rather notoriously bad piece of apparatus at times. It may go into the pelvis, and there have been cases in which a bowel and a bladder have been punctured. It has also appeared penetrating the gluteus muscle.

The selection of the nail, of course, is basic. In the event that the nail selected is too short, there are some projections now that are used to attach to the end of a nail made up in ¼, ½ and ¾ inch lengths, and so on, that can be added to it.

The introduction of a Smith-Petersen nail without the use of a guide pin is hazardous. Reinsertions are sometimes required. These are damaging.

We have devised a counter-bore in our group which we would like to recommend to those of you who have never seen or used one. This piece of apparatus is threaded over the guide pin, and a hole is made in the cortex of the femur to make a "set" for the insertion of the Smith-Petersen nail. We have found this very advantageous in intertrochanteric fractures so as not to further traumatize the area of the femur at the base. These additional fractures have also been produced by those who have nailed a simple femoral neck fracture.

I would like to ask Dr. Maibauer if at some future time he would let us know about that last slide, in which an osteotomy was done. The femoral head looked sclerosed.

The insertion of a nail over a guide pin, done in

the early days, was with the use of a Kirchner wire, which bent very easily and presented difficulties. The pins, now, are so constructed as to give resistance. We must not forget however, that small fragments of bone can sometimes get into the canal of the Smith-Petersen nail, and the guide pin can then traverse into the pelvis, as a result of that obstruction.

Two cases recently gave us some trouble, in which we improperly selected the length of the nail for an intertrochanteric fracture, and it penetrated only to the base of the femoral head. These two particular patients had a subsequent injury, and had a femoral neck fracture occur beyond the nail insertion. We were then obliged to remove the nail and its plate attachment, insert a longer nail and re-attach the plate. I would like to recommend that the nail be driven into the body of the femoral head for some distance as a protection against this hazard.

I. B. Harris (Detroit, Mich.): I do not think there is any better way for us to learn something about this problem than to review a series of cases such as Dr. Maibauer has over a period of ten years. In this way we radiologists can learn how we may detect some pitfalls, and surgeons can learn a few of their mistakes.

I think as far as procedure is concerned "every man to his own tools." I happen to have had the opportunity to view other technics in fixation of hips. One man can do it one way better than another, and vice versa. A simplified technic such as this one, to my way of looking at it, is best. A great many of us do not have complicated apparatus, and a special operating room to do this type of procedure, so we must use the tools we have.

From the radiologist's point of view a little patience on the surgeon's part in allowing us to get the apparatus ready is essential. The most important thing, of course, is the accurate centering of the x-ray machine. It must be directly over the fracture in the anteroposterior view, and it must be in direct parallel in the lateral view. If you will elevate the hips sufficiently so that the x-ray tube can get down to the level with the fracture, the distortion which frequently causes misjudgment can be eliminated.

I need not go into all the technics of reduction, but from observing a large series of cases I think failure to maintain internal rotation after reduction is a most important thing. If there is internal rotation to such a degree that the lesser trochanter cannot be visualized on an x-ray film, we have the best opportunity to see the neck of the femur in the lateral projection. For some reason or other it also seems to reduce the tendency of the pin to go up or down, or angulate in an anteroposterior plane, which is frequently the biggest bugaboo as far as what happens after the injury.

One complication we noticed in quite a few cases, I think about eighteen, was intrusion of the nail. One can give it a chance to see if there is healing despite this fact. It has been our observation, however, that if there is an extrusion of the nail, one might as well give it up and start over.

Finally, I would also like to say that if, after the initial postoperative examination made in the x-ray department where the technic can be selected, the reduction is not good and the insertion is not within reason, one might as well take the embarrassment then as later.

There is one other thing that I would like to point out and that is the dangers of this procedure. A lot of the younger men in particular are a bit brash about exposure to x-ray. It has just recently been pointed out that in taking the lateral projection alone, the scattered radiation to the nursing staff, and to the technician, or to the doctor who usually holds the film, is about 300 milliroentgens, which is three times a daily tolerance dose, as far as you gentlemen are concerned. If you do a lot of these, in time it is going to count. So I would certainly advise you to alternate the people that are going to hold the film, and get your patient in as perfect a position as possible, to begin with, so that you do not have to take repeated x-rays to make sure of your position at the time of surgery.

Gordon Mackay Morrison (Boston, Mass.): I would like to say a word about Dr. Speed's article and Dr. Ghormley's and ask them their advice.

Dr. Speed's cases were most unusual. I do not recall ever having seen a bilateral dislocation of both hips. Speaking of unusual cases recently I saw a six year old child with a complete posterior dislocation of the right hip. He sustained the injury while playing on a log with some other child who pushed him. Now, that's the youngest one I have seen.

At the Boston City Hospital I have had the assignment for the past year of posterior hip dislocation, and a former speaker here, Dr. Childress, remarked that he had used the Gibson approach. I have used it in three cases and I like it very much. It gives a good approach.

A year or so ago when Dr. Lorenz Boehler was here from Vienna, I asked him what he did on his straight dislocated hips with regard to weight-bearing. That has always been a boogie to me and still is. He shrugged that one off rather quickly and said, "I let them walk in three or four weeks." I said, "How many have you seen, sir?" He said, "Oh, 40 or 50."

That made me feel that I was being a little too conservative, perhaps, in keeping them off weight-bearing a number of months and checking with x-ray periodically.

From Dr. Speed and Dr. Ghormley, what I would

like to know is, after the straight ordinary dislocation of the hip, how long should we keep them off weight-bearing?

MARSHALL R. URIST (Los Angeles, Calif.): The end results of traumatic dislocation of the hip joint have been surveyed by nearly every large surgical clinic in the world. Each report supplies valuable data. Dr. Ghormley and Dr. Speed have just brought the subject up-to-date and we have now heard a summary of the problem by two of the most experienced surgeons of our time. I would like to take this opportunity to ask Dr. Speed and Dr. Ghormley a question which may have some bearing upon the etiology of avascular necrosis of the head of the femur. This complication has been described following posterior dislocations, central fracture-dislocations, and even in two cases after an isolated fracture of the greater trochanter seen by Quist-Hansen. Have either of you gentlemen seen or read of a case of avascular necrosis following an anterior dislocation of the hip joint? The superior portion of the head of the femur is supplied by blood vessels which cross the epiphyseal line in the posterior reflected capsule and the retinacula of Weitbrecht. Two patients upon whom we operated to replace a large fracture fragment of the posterior rim of the acetabulum showed subchondral compression fractures of the head of the femur and thrombosis of the retinacular blood vessels. These observations suggest that avascular necrosis originates with trauma which is primarily posterior in location and occurs at the time of the original injury. It would not, thus be influenced appreciably by such treatment as traction, restricted weight-bearing or immobilization. It is conceivable that the intra-articular structures located posteriorly can be injured in an anterior dislocation, but the late complications, for various reasons, are less common and much less serious in anterior dislocations than in posterior dislocations.

FRANK P. STRICKLER (Louisville, Ky.): Personally I have never seen a bilateral dislocation of the hip. This audience presents a pretty good cross section of the country. I would say we have some experienced men here who have seen it.

Next I want to say that in nailing hips, and I have nailed quite a few of them having started back in the old days with Whitman and Albe, that the first principle in nailing a hip is to reduce it. The next thing is to hold it reduced, and the only way that I have been able to do that is on a Halle table, or a modification of one.

You need never have any fear about the rotation of the head of the femur because if it rotates, simply put the hip up in the Whitman position of extreme abduction and with internal rotation that locks the head in position it is very simple to nail.

We talk about these wires. There is one sure way of avoiding trouble with guide wires, and that is not to use them. Most surgeons seem to have lost sight of the fact that they have an index finger that they can feel all around the neck of the femur with, if they want to. I have nailed between 400 and 500 hips, most of them fairly successfully, and I have never used a guide wire. I do not think they are necessary at all, and we nail hips in anywhere from thirty to forty-five minutes.

I think in these elderly patients time is very essential, because they are on the downhill side of life, and to put them on the operating table and keep them there three or four hours and give them 4 or 5 pints of blood in my opinion is absolutely inexcusable.

OSCAR PERRY HAMPTON, JR. (St. Louis, Mo.): It seems to me that this discussion of dislocations of the hip should not go by without some emphasis on the dashboard as the strong etiologic agent. In our experience dashboard dislocations are the most common type.

Dr. Speed did not have a drawing of that type, in which the knees are thrown forward against the dashboard, the acetabulum is broken out posteriorly and the head of the femur goes posteriorly. My one case of bilateral dislocation of the hips resulted from simultaneous dashboard impacts.

I mention this mechanism of injury for another reason. Dr. Ghormley emphasized the importance of early diagnosis in the prevention of complications. Too often a posterior dislocation of the hip is overlooked because of an obvious knee injury due to the impact against the dashboard. It may be a fracture of the patella; for example a house officer calls stating that a closed fracture (note, Dr. Kennedy, I said closed and not compound) of the patella, is being admitted. The dislocated hip is completely overlooked. Particularly, it may be overlooked if there is an open fracture of the patella, to which attention is obviously directed.

If one will always keep in mind that in every dashboard accident there may be a fracture of the acetabulum, and a posterior dislocation of the hip, or even a dislocation without fracture, fewer will go unrecognized.

ROBERT P. DOBBIE (Buffalo, N. Y.): My comments are directed toward a remark of Dr. Speed's. I hope he will be as tolerant of my relative inexperience and ignorance as I am respectful of his great ability, and, more important, the fact that Dr. Strickler brought out, that when Dr. Speed speaks, it has weight behind it.

He mentioned as late sequelae of dislocations of the hip femoral hernia, inguinal hernia and hydrocele. I think it is important, before that gets into print, to make sure that it has any relation to a dislocation of the femur. It is a surprise to me. I have never seen hip dislocation listed as a contributory etiologic factor in hernia.

We have a lot of trouble in compensation courts

with hernia alleged to result from everything from head injuries to toe injuries, and I would like Dr. Speed to elaborate a little in order to try to persuade me that there is any relationship whatsoever to these hernias demonstrated late after hip dislocation.

F. P. MAIBAUER (closing): I want to thank Dr. Curry for his additional remarks, in that these are really treated as closed, because we do not in fractures of the neck expose the fracture line.

We do always use a burr before insertion of the nail to prevent splinting of the neck of the femur, or the greater trochanter in intertrochanteric fractures. As stated in the article we do use a guide wire, because we believe, in our hands, that we are better able to place the nail.

KELLOGG SPEED (closing): To answer Dr. Dobbie first, because I cannot quite hold my breath in the matter, I would say that we came here to be surprised. There is no doubt that these conditions of hydrocele and hernia have existed following traumatic dislocation of the hip; and if he will read my article when it is published and study the references, he will find that some of the men as long as fifty and sixty years ago found such things.

The patient I had reported upon I had known for years. He never had the slightest evidence of a hernia. I had examined him on more than one occasion, and this came on some five years later. When I opened the sac, it was a very small sac, rather thick—I will admit that—probably congenital, and without the usual amount of fat around it, with a narrow opening into the peritoneal cavity.

But it is not difficult to conceive that the trauma of the dislocation, the force and pressure against the abdomen, or the hip, or the whole body, might enlarge or weaken the arch of the internal ring, or the femoral ring, and cause some weakening and spreading of an already pre-existing small glove-finger typed sac, which would lead to a hernia showing up some time later, maybe only after a few years.

To answer Dr. Morrison, I would suggest eight weeks of weight-bearing freedom; but the patient in bed or in a wheel chair may, after two weeks. use active motions of the hip.

Dr. Urist's remarks were very kindly. I have never seen an aseptic necrosis of the head of the femur following an anterior dislocation, and anterior dislocations are not so very common. It is my idea, coupled with what the pathologic specimens which were displayed showed, that a great deal of this may be due to two factors: first, pressure from the clots in the misplaced head, and so on; and second, and maybe equally important, the thrombosis existing in the vessels thereabouts, rather than complete severance of all the blood supply, even that from the ligamentum teres.

I must ask you for your forebearance because Dr. Ghormley did not mention the fact that Mr. Nicoll at the meeting in London in June reported "144 cases of traumatic dislocation of the hip, and the incidence of aseptic necrosis at the head of the femur was 10 per cent. If the incidence reported from other sources varied, this was because necrosis was often confused with traumatic arthritis. The two conditions were very different. Necrosis was a vicious process; radiographic evidence, always present within two years of injury, preceded clinical features. Traumatic arthritis was a common, relatively benign complication of later development and slower course. Necrosis was commoner in children than in adults because there was absolutely no anastomosis across the epiphyseal cartilage-plate to make up for the interference with the blood supply to the femoral head caused by retinacular damage." I would rather be inclined to dispute that statement.

"It might therefore be reasonable to carry out prophylactic drilling of the cartilage plate after dislocation in childhood, but if so there was no point in blocking the new channels with bone grafts. Half the cases of necrosis were associated with delayed reduction of repeated attempts at reduction, so early recognition and gentle management were obviously essential. Premature weight-bearing was also dangerous; 20 per cent of patients allowed to take weight within a month of injury developed necrosis, whereas the incidence was only 7½ per cent when walking was delayed. But even prolonged abstention from weight-bearing would not facilitate reconstruction of a normal head, once necrosis was established, by creeping substitution; the revivication process was too slow to prevent cartilage degeneration and massive collapse. The over-all incidence of necrosis was not high enough to justify routine prohibition of weight-bearing for more than eight weeks, but every case must be examined radiographically every three months for two years. If radiographic evidence were seen, management was a difficult problem, for disability might be trivial dispite the most alarming appearances. Of 15 cases of necrosis, only 6 required operation (2 arthrodeses and 4 displacement osteotomies); the remaining patients had trivial symptoms and were all working."

RALPH K. GHORMLEY (closing): In answer to Dr. Morrison's question about the time weight-bearing is prohibited, we try to keep our patients in bed four weeks after reduction of simple dislocations and on crutches for two additional months.

The patients do not always follow this program, I am sure, because many of them get along well and do not feel that this is necessary. That is arbitrary, I admit, and I do not think we know the exact

time weight-bearing should be prohibited. We have not observed any instance of aseptic necrosis after anterior dislocation in our series of cases.

After looking at Dr. Urist's slides and listening to his remarks, I think that it has been emphasized that we do not know the extent of the damage and the pathologic change in many cases of fracture of the acetabulum and dislocation. Until more open reductions are carefully performed and a study of the tissues is made at the time of the operation, we will not know the full extent of the injury. Speed and Stewart emphasized that the capsule was very important. The ligamentum teres, the fossa acetabulae from which the ligament passes, and other structures probably are injured. Our knowledge of these important pathologic changes is not sufficient to permit us to piece together the whole picture, which I hope eventually will be known.

# SECONDARY TENORRHAPHIES AND TENDON GRAFTS IN INJURIES TO THE HAND

JOSEPH L. POSCH, M.D.
*Detroit, Michigan*

IN the surgery of trauma the incidence of injuries to the hand is remarkably high. It has been estimated that one-third of all industrial injuries involve the upper extremity. In fact, in some industrial areas hand accidents comprise one-half of all injuries. The period of disability is often longer than that required for so-called more serious accidents such as fractures of the long bones and back injuries. It is well known that in a severe hand injury the patient's entire future economic status may be seriously hampered by decreased earning power.

Fortunately, many of the injuries are superficial and respond well to simple suturing. On the other hand, many of the accidents involve tendons and nerves which require a great deal of care and definitive work, and it is only by following basic principles[1–4] emphasized repeatedly that good results can be obtained.

Tendons of the hand may be injured in several ways. The most common cause is by direct trauma with severance of the skin and underlying tendons. This injury may vary from a laceration 1 cm. in size to a large avulsed wound involving not only tendons but also large areas of skin, nerves, joints and bones. Tendons may be ruptured by severe external violence with a non-penetrating injury, or they may rupture following fractures. Infection, either specific or non-specific, likewise can lead to destruction of the normal continuity of the tendon.[5] This infection may be due to tenosynovitis or it may be a complication of an unsuccessful primary repair.

*Diagnosis.* The diagnosis of flexor tendon injuries is extremely important. One frequently sees very small lacerations that have resulted in severed flexor tendons. (Figs. 1 and 2.) The patient usually thinks that only a superficial cut is present but on resuming activity he frequently finds that motion is impaired.

The proper diagnosis of these injuries cannot be emphasized enough. The wound need not, and should not, be probed to establish that the tendon has been severed. Physiologic tests can readily identify any serious injury. Inability to flex the distal phalanx signifies that a flexor digitorum profundus tendon has been severed, while inability to flex the entire finger is proof that both the flexor digitorum profundus and the flexor digitorum sublimis tendons have been injured. Loss of flexion of the fingers at the metacarpophalangeal joint, with inability to extend the distal two finger joints, reveals injury to the lumbricales or interossei. Frequently a tendon may only have been partially severed. Routine tests at the preliminary examination will reveal good function only to have complete separation occur at a later date when motion is resumed. To avoid this needlessly embarrassing situation, the tendon should be flexed against pressure at the original examination. Inability to flex the distal phalanx of the thumb is evidence of a lacerated flexor pollicis longus tendon.

At the time of the original examination the injuries to the nerves of the hand must also be considered. Lacerated digital nerves are associated with a resultant loss of sensation in the involved fingers. More proximal in the wrist, injuries to the median and ulnar nerves are shown by anesthesia over the palmar aspect of the fingers. For example, a lacerated median nerve results in loss of sensation over the thumb, index, long finger and radial side of the ring finger. A severed ulnar nerve results in loss of sensation over the little and ulnar side of the ring finger. These tests may be substantiated by testing the intrinsic muscles of the hand.

The question whether primary or secondary tenorrhaphies should be carried out has been well discussed frequently by others. In general, flexor tendons of the fingers should not be primarily repaired if the injury is more than several hours old. This is especially true if there has been any probing of the wound, ligating of

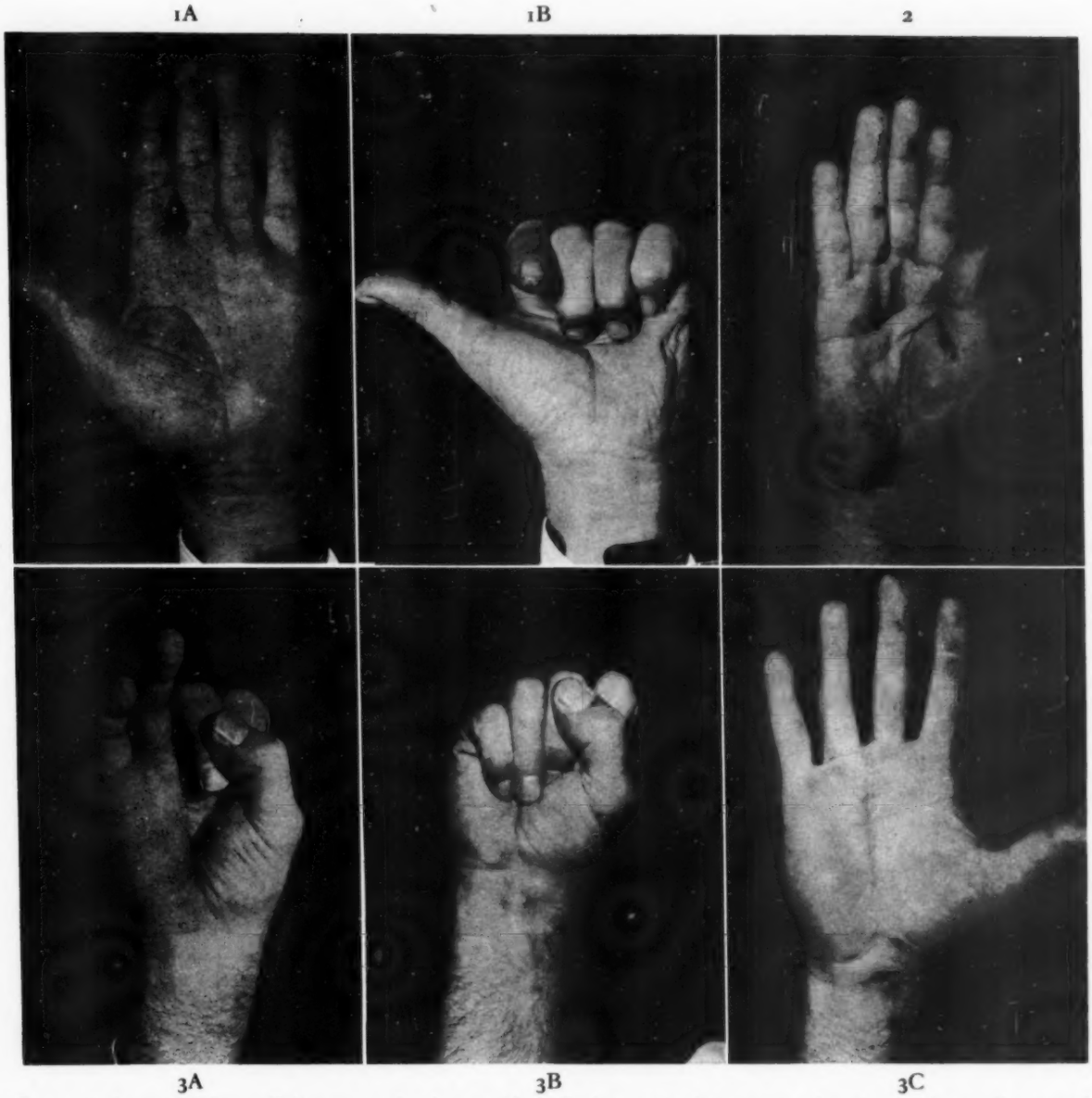



FIG. 1. A, lacerated flexor digitorum profundus, accidentally incurred when cut on a hunting knife; small external wound is visible in proximal crease of finger. B, note inability to flex distal phalanx.

FIG. 2. Lacerated flexor pollicis longus through small laceration.

FIG. 3. A, open fracture fourth metacarpal, with severance of flexor digitorum profundus tendon to ring finger and injury to tendons of little finger. B, after healing of fracture; tendon graft of profundus in palm and tendolysis of flexor tendons to little finger carried out. Good functional result obtained. C, extension postoperatively; the incision in the palm was the site of the original accident.

vessels or an attempt at primary repair. In addition, repair should not be done when fractures accompany the injury. (Fig. 3.)

Secondary repair may be carried out as soon as the skin is well healed. This is usually after three or four weeks. If the tissues have not healed well and the scar is red and indurated, secondary procedures should be delayed up to two months. Of course, if a frank infection is present, the procedure must be delayed until all signs of infection have subsided. After that, tendon grafting is usually needed since contracture of the muscle belly has occurred and it is impossible to stretch the muscle so that the ends may be united. It has been our experience, in general, that in the finger tendon grafts

FIG. 4. Sutured digital nerve; tendon graft (extensor of foot) is placed with paretenon about it.

do better than do secondary tenorrhaphies, and in the palm and wrist secondary tenorrhaphy is feasible, even after several months.

Candidates suitable for reconstructive surgery of the hand must present good skin, good joints and good sensation. In addition, the more cooperative and eager the patient is to get a good hand, the more likely he is to obtain one that is functional and useful.

Good skin is extremely essential; and if scarring and contracture are present, a pedicle graft must be applied. Passive joint motion is extremely important, for it is impossible to conceive that a tendon graft will flex the joint actively to any greater degree than could have been done passively preoperatively. The condition of joints in the finger also depends a good deal on the age of the patient. In tendon grafting young adults obtain better results than older individuals whose joints have a definite tendency to stiffen in even relatively short periods of immobilization.

Sensation is important. Digital nerves can be repaired at the time of the primary wound closure. We have not done secondary digital neurorrhaphies, then with the return of sensation, carried out tendon grafting. Usually, digital nerves are repaired at the same time as the tendon work is done. (Fig. 4.)

If severe injury has occurred to the finger with severance of both tendons, entire blood supply and nerves, the patient may not be a

FIG. 5. A, loss of flexion of long and ring fingers following severance of flexor digitorum profundus over the middle phalanx.

good choice for reconstruction. Graham calls this "local shock." Secondary atrophy occurs; later a large ridge on the nail, signifying the duration since the time of the accident, may also be present.[6]

Often in severe extensive injuries to the finger, amputation rather than tendon grafting should be considered. It has been stated that tendon grafting of the little finger, especially of the profundus, should not be done unless the individual is a typist or pianist. We, however, have performed grafts on all individuals presenting old lacerations of the tendons of the little finger; and although it is true that they are frequently unable to flex the distal phalanx completely, they do have increased strength and are able to hold objects well. Graham[8] and Littler[7] recommend that in a lacerated flexor digitorum profundus with an intact sublimis the distal phalanx be ankylosed at 30 degrees of flexion. The patient will then be able to bring the finger tip well into the palm.

In these reconstructions of tendons in the fingers we are always faced with the problem of whether to do secondary tenorrhaphies or tendon grafts. Many times it appears to be more feasible to do an end-to-end suture, even

5B 5C

FIG. 5. B, flexion obtained by "advancement" of deep tendons to the distal phalanx. C, limitation of extension of fingers due to shortening of tendons. However, the patient, a carpenter, has a good functioning hand and does all of his work.

in "no-man's land" (the area between the distal flexion crease of the palm and the distal phalanx). However, in our series of cases better results are obtained with tendon grafts. We have on several occasions "advanced" the flexor digitorum profundus to the distal phalanx; and although there is some shortening, good functional fingers have been obtained. (Fig. 5.)

## OPERATIVE TECHNIC

Once reconstructive surgery has been decided upon, certain basic principles should be followed. Thorough preoperative examination, of course, should include complete physical examination, blood count, urinalysis and so forth.

Sterile preparation of the operative site the night before, with thorough washing with soap and water on the day of surgery is important.

*Bloodless Field.* These surgical procedures are preferably carried out in a bloodless field. This is obtained by the employment of a blood pressure cuff as a pneumatic tourniquet. Sheet wadding is placed on the upper arm, the blood pressure cuff is placed over this and the cuff is maintained in place by roller gauze. The arm is elevated for sixty seconds and the blood pressure cuff is rapidly inflated to 280 mm. of mercury. If necessary clamps may be placed on the tubing next to the cuff itself, but frequently this is not necessary. The cuff is left on for one hour and a half, and is then removed. The bleeding vessels, if not already tied, are clamped and ligated with fine cotton.

*Instruments.* Small instruments, of course, are extremely important; No. 15 knife blades, fine tissue forceps and small forceps are essential to gentle handling of tissues. A curved, double-edged scalpel as suggested by Koch[9] is helpful in freeing tendons; as are the tendon strippers and tendon leaders of Bunnell.[10]

*Incisions.* Incisions in the hand follow the flexion creases. In the finger the incision is made in the middle lateral position. The small digital nerves and vessels are retracted anteriorly. In the palm incisions follow the flexion creases. Transverse incisions are made in the wrist except when the palmaris longus tendon is being removed; then a longitudinal incision is made the length of the tendon.

All scar tissue is carefully excised and the extent of the original injury determined. In the finger where the profundus has been separated and the sublimis is intact, not scarred and is functioning, we do not remove it although this has been done in just such instances with favorable results.[11]

*Sources.* Common sources of tendons for grafting are the flexor digitorum sublimis, the palmaris longus and the long extensors of the second, third and fourth toes. In our work

FIG. 6. A, disturbance in continuity of both flexor tendons in long finger when cut by glass. B and C, flexion obtained after tendon graft (extensor of toe) to middle finger, and tendolysis of flexor tendon to ring finger. The graft appears to be a little long but the patient has a good functioning hand and does not want a tendon-shortening procedure done. D, extension.

we prefer the extensor tendons of the toes. The tendon itself is small in diameter, whereas the sublimis is much larger and does not, of course, have the surrounding gliding material. The tendon in the foot is removed through a semicurved, longitudinal incision. If a sublimis tendon is used, subcutaneous fat is often removed from the abdomen and placed about the tendon to aid in gliding. The palmaris longus tendon may be utilized. We have been reluctant to make the rather long longitudinal incision required on the forearm when the extensor tendons of the foot work so well. We have seen no difficulty from removal of these tendons.

In flexor tendon injuries of the fingers the scar tissue must all be carefully excised and the tendon sheath opened. The pulleys over the middle phalanx and proximal phalanx may be preserved if they are intact and if the tendon graft is small enough to glide through them safely. The technic of tendon grafting which we have recently adopted is that described by Allen.[12] This consists of suturing one end of the tendon graft to the distal phalanx. Bone is

FIG. 7. Passing tendon graft from finger to palm; note intact sublimis and opened pulley.

exposed; the graft is held in place by suturing it to the periosteum and also to the remaining portions of the profundus tendon itself. The tendon is then placed beneath the pulley. If the pulleys have been destroyed, others are reconstructed with small portions of tendons sutured over the former sites of the pulleys or placed completely around the finger under the extensor tendon. We often use the distal insertion of the sublimis tendons for pulleys. This can be done by crossing the slips to the opposite side of the phalanx. One must be careful not to cut these sublimis insertions too long so that they become attached to the tendon graft, or too short so that hyperextension of the joint occurs. Hyperextension of the proximal interphalangeal joint occurs frequently in tendon grafting, and according to Graham[8] it can be corrected by attaching the distal portion of the sublimis tendon across the joint to the proximal phalanx in 10 degrees of flexion.

Determination of the length of the tendon is extremely important. In healing there is definite shortening of tendon grafts. In three of our postoperative cases, two involving the little finger and one involving the ring finger, reconstructive surgery consisting of tendon lengthening had to be done because of extreme shortening. In another case (Fig. 6) the tendon function, although good, was hampered because the tendon itself was a little too long. In our opinion, the extensor tendons of the toe contract more than do sublimis tendon grafts. We have found that with use of the sublimis tendon best results were obtained when the wrist was flexed, as recommended by Koch,[10] to an angle of 110 to 120 degrees and the metacarpophalangeal joint was flexed to 120 degrees. The ends are then united at normal tension. In tendon grafts that are obtained from the foot we have used Bunnell's method[11] in which he suggests keeping the wrist and fingers almost straight out and suturing the tendons at a slight degree of tension.

The tendon graft is then brought into the incision in the palm by means of a tendon leader. (Fig. 7.) Suture is accomplished as far proximal in the palm as possible. Since there is a rather large disproportion between the graft and the flexor digitorum profundus, rather than attempt to unite the tendon ends by direct suture we have adopted Allen's[12] procedure. This consists of making a small longitudinal slit in the tendon, threading the tendon through it and then suturing them side by side. In addition, we have beveled the profundus tendon and sutured this to the tendon graft. If the flexor digitorum sublimis has been removed, the proximal end may then be sutured

8A 8B 8C 8D

FIG. 8. Exercises useful in restoration of hand function. A, rubber ball, although extremely popular, is good only for action on the metacarpophalangeal joint. B, wooden block is excellent for tendon motion. C, small objects work well for finer movements. D, warm, soapy hand baths along with squeezing a rubber sponge aid in restoring function.

to the profundus tendon. If at all possible the lumbrical muscle is placed over the site of the union.

The digital nerves should be sutured at the time of repair. No. 6-0 eye silk is used for nerves. The suture material that we have employed for tendon procedures has been No. 60 cotton on small, curved cutting needles.

The skin edges are closed either with No. 60 cotton or 5-0 dermalon, and several tiny rubber drains are placed in each incision and removed at the end of twenty-four hours. A rather large pressure dressing with a posterior splint is then applied. The wrist and fingers are placed in moderate flexion. Most of the relaxation is obtained at the wrist while the fingers are left fairly straight.

In lacerations involving the thumb, with severance of the flexor pollicis longus, the foregoing procedures for the fingers apply also. The tendon graft should extend from the distal phalanx to the wrist.

Immediately postoperatively the extremities are kept elevated on several pillows and the patient wears a sling when he is up and about. He is kept in the hospital for three to seven days, depending a great deal on the individual and his reaction to the procedure.

Sutures are removed in one week to ten days and the supporting splint is worn for four weeks. A little early motion is encouraged but not insisted upon. At the end of three weeks the patient is asked to remove his hand and arm from the cast and bathe it in warm soapy water

once a day. He continues to wear the splint at night and when it is not being soaked. At the end of four weeks the splint is removed and he is asked to wash his hand in warm soapy water for twenty to thirty minutes, three times daily. At the end of that time active motion is started. Active exercise consists of squeezing of a rubber sponge in water, the employment of wooden blocks and occupational therapy. (Fig. 8.)

At the end of six to eight weeks many of these patients can return to work.

### CASE REPORTS

The following is a case report of a patient with (as Boyes[13] describes) multiple operations, with ill advised median longitudinal incisions plus subsequent infection. This resulted in severe scarring (Fig. 9) so that a pedicle graft was necessary.

CASE I. A. L., a twenty-seven year old plant administrator, suffered a severe injury of his right hand on October 26, 1948, when it was cut by a sharp, heavy block. The middle, ring and little fingers were lacerated. The little finger suffered a very slight injury. In the case of the ring and middle fingers, however, there apparently was division of the flexor tendon.

On December 17, 1948, the fingers were opened and a tendon repair done. The patient stated that a kangaroo tendon repair was the procedure employed and that the fingers were placed in plastic tubes to keep them in position. The fingers were kept in these splints for four days, after which the patient was instructed to soak the hand every day. At the end of three weeks there was no motion in the fingers at all. The hand was then reopened in January, 1949, and holes were drilled to the middle phalanges. Tendon ends were transplanted in these openings.

When first seen in September, 1949, examination revealed irregular longitudinal scars over the volar surfaces of the right middle and ring fingers, extending from the distal interphalangeal joint proximalward into the palm to the region of the distal transverse palmar crease. These scars were contracted, the ring finger moderately and the middle finger more severely. On October 10, 1949, small pedicle grafts were applied to the right middle and ring fingers after excision of the scar tissue. This pedicle was taken down on October 29th.

On February 6, 1950, employing the flexor digitorum sublimis tendon, tendon grafts were made from the proximal portion of the profundus tendons in the palm to the stumps of the tendons in the distal phalanges. Pulleys were made over the proximal phalanx by means of small portions of extensor tendons taken from the toes. The patient's postoperative course was uneventful and at the end of four weeks motion was started. The tendon to the long finger functioned very well. However, on December 20th it was necessary to perform a tendolysis and a tendon-lengthening procedure to the tendon of the ring finger.

On January 15, 1951, the patient was activated from the Reserves and accepted for full duty with the Air Corps. Although there is a flexion deformity of the distal interphalangeal joint of the ring finger and hyperextension of the proximal interphalangeal joint of the same finger, he has a good functioning hand and is able to perform all of his work well. (Fig. 9D and E.) (This hyperextension of the proximal interphalangeal joint was present before the reconstructive work began and probably could have been corrected as Graham points out, by flexing the joint in 10 degree flexion and uniting the sublimis tendon to the proximal phalanx.)

The following is an example of a ruptured flexor pollicis longus corrected by tendon graft:

CASE II. Patient O. K. K. was first seen on February 29, 1952, with a history of an injury to his right thumb about five days previously. He stated that while tightening a wing nut on a clamp he felt something move in his thumb, after which he was unable to bend it. Examination revealed inability to flex the thumb. (Fig. 10A.) Sensation was intact and good passive motion of the joints was present.

The patient was admitted to the hospital and the operative report is as follows:

"Anesthesia was satisfactorily obtained by means of sodium pentothal and nitrous oxide. The entire left hand was throughly washed with soap and water for ten minutes. Ether and alcohol were then applied. Sterile drapes were placed in the usual manner and the arm was extended along an arm board.

"A midlateral incision was made on the lateral aspect of the thumb and the flexor pollicis longus tendon was identified. It was found to have narrowed and to have ruptured about the midportion of the thenar eminence. (Fig. 10B.) The distal portion was brought out through the incision in the thumb itself.

"A transverse incision was then made in the wrist and the flexor pollicis longus was identified here. This was done with some difficulty because the flexor pollicis longus tendon had become quite adherent and it was difficult to distinguish it from the flexor digitorum profundus to the index finger. However, by enlarging the incision proximalward along the radial side of the wrist for about

9A 9B

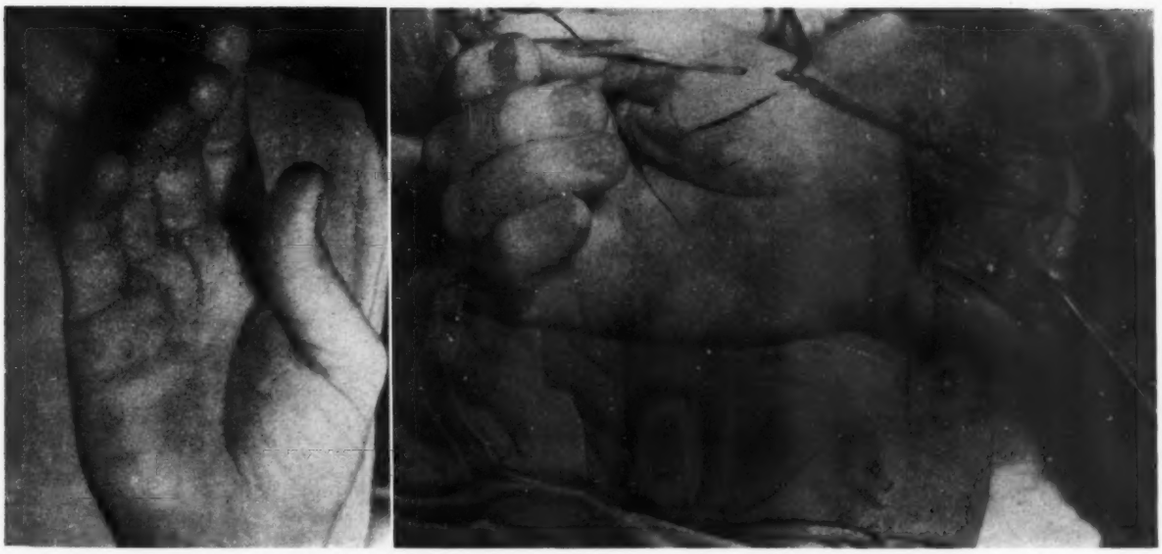
FIG. 9. A, inability to flex middle and ring fingers following severance of profundus tendons and "multiple" operations. B, limitation of extension because of midline incisions and infection.

9C 9D 9E

FIG. 9. C, replacement of cicatrix by pedicle grafts. D, flexion following tendon grafts. (Sublimis tendons used as grafts.) E, extension possible following reconstructive surgery.

7 cm. the origin of the flexor pollicis longus was identified.

"A small transverse incision was then made at about the midportion of the thenar eminence and the flexor tendon sheath of the flexor pollicis longus was identified here. By means of tendon strippers the proximal portion of the flexor pollicis longus tendon was freed from scar tissue and brought out in the wrist. It was noted that through some diseased process the flexor pollicis longus tendon had gradually atrophied and eventually ruptured at about the midportion of its course over the midportion of the thenar eminence. Since it was thought that even with suturing the tendon would not be strong enough for a good result, it was decided to do a tendon graft. Accordingly, the long extensor of the 3rd toe of the right foot was removed for approximately 7 inches



10A 10B

FIG. 10. A, loss of flexor power of thumb due to rupture of flexor pollicis longus. B, findings at surgery; note thin frayed ends of tendon at site of separation.

and then sutured to the distal portion of the flexor pollicis longus tendon in the thenar eminence and to the proximal portion of the tendon in the wrist. This was accomplished first by suturing it on the wrist and then tunneling it by means of tendon leaders into the thenar eminence. Here it was then also approximated very carefully end to end. This was done with interrupted No. 60 cotton sutures. The wound was thoroughly irrigated several times during the procedure. The bleeding vessels were controlled with pressure and ligation and the skin edges were then closed with interrupted and continuous 5-0 dermalon sutures. Several small rubber drains were inserted in each operative site for drainage. The foot was likewise closed with interrupted and continuous 5-0 dermalon sutures and a pressure dressing was applied.

"The hand was dressed in a moderately large pressure dressing with flexion obtained at the wrist. The foot was dressed with a small pressure dressing."

The microscopic diagnosis of the ruptured portion of the tendon that was sent to the laboratory was chronic synovitis of tendon.

The patient's postoperative course was uneventful and he was discharged from the hospital one week after the operation was performed. The posterior splint was removed at the end of four weeks, and at first motion was very slow. However, he gradually received strength in the end of his thumb. When last seen he was able to use it for writing and in all of his daily work. (Fig. 10C.)

Case III is an example of a severe crushing injury to the fingers with severance of tendons and nerves.

FIG. 10. C, flexion following tendon graft (extensor of toe).

CASE III. J. S., a forty-eight year old machinist, suffered a severe laceration of his left index and middle fingers while working on a machine that faced bearings. The machine tripped and a weight

11A 11B

FIG. 11. A, crushing injury of fingers with severance of tendons and nerves to index and middle fingers. B, extension.

11C 11D 11E

FIG. 11. C, flexion following tendon grafts (sublimis tendons). D, good range of motion after tendolysis, tendon shortening and placing of fat about tendon graft to index finger. E, extension is practically normal; the ring serves as a pulley.

shuttle fell down and severely ripped the left index and middle fingers. Primary repair was carried out on the flexor tendon. Healing occurred without infection. He had previously injured the tips of his left index and middle fingers in September, 1939.

When first seen examination revealed healing transverse lacerations of the palmar surfaces of the proximal phalanx of the index and middle fingers. There was definite limitation of motion. On active motion the tip of the index and middle fingers could be brought only to within 4 inches of the palm. (Fig. 11A.) Passively they could be brought only to within 3 inches of the palm. The patient was told to bathe his hand in warm soapy water several times daily. Passive motion was obtained in the joints and the indurated skin became soft and pliable.

On February 23, 1951, he was admitted to the hospital and under general anesthesia tendon-grafting procedures were done. The operative report is as follows:

"The hand was thoroughly washed with soap and water; ether and alcohol were then applied. Sterile drapes were applied in the usual manner and a blood pressure cuff was applied and used as a

12A

12B

12C

12D

FIG. 12. A, lacerated flexor digitorum profundus to ring and little fingers; flexion possible preoperatively. B, extension preoperatively. C, flexion obtainable postoperatively, following the insertion of tendon grafts (extensors of toes). (Technic described by Allen.) D, extension postoperatively.

tourniquet. A midlateral incision was made on the radial aspect of the index finger. This incision extended from the midportion of the distal phalanx proximally to the base of the finger and then along the flexion crease at the thenar eminence. The skin flap was retracted. The digital nerves and vessels were identified. The flexor tendon was found to be united by black silk sutures but was bound down in a large amount of severe scar tissue. The sublimis tendon had been partially removed. It was necessary to remove the flexor tendon throughout its entire length because of the severe scarring. A midlateral incision was then made on the radial aspect of the middle finger. The digital vessels and nerves were identified. The nerves were found to have been cut and separated quite widely as they were in the index finger. Again in this finger the flexor tendon was found to be united with black silk suture but was again bound down in a good deal of scar tissue and the ends of the sublimis tendon were united to this scar and to the deep tendon.

"In order to visualize the digital nerve better a small transverse incision was made just proximal to the base of the middle finger. A transverse incision was then made in the wrist and the sublimis tendons were removed for their entire remaining length. Employing them as tendon grafts the

distal portion of the profundus tendon in the tip of the finger and the proximal portion of the profundus tendons in the palm were united. The digital nerves in both fingers were thoroughly freed of all scar tissue, all neuromata excised, and the freshened ends were approximated with 6-0 silk sutures. The tendons were approximated with 5-0 cotton.

"The wound edges were closed with continuous 5-0 dermalon sutures. The hand was dressed in a modified pressure dressing with the wrist and the metacarpophalangeal joints flexed to 110 to 120 degrees. A posterior plaster splint was applied and the dressing was reinforced with elastic bandages.

"During the operation the patient received 2000 cc. of 5% glucose and distilled water."

The tissues healed without infection and the patient returned to work in June, 1951. However, although motion in the middle finger was good, he had limitation of motion of the index finger. Consequently, he was readmitted to the hospital on October 20, 1951, and tendolysis was done of the flexor tendons of the middle finger; tendolysis and tendon shortening was done of the flexor tendon to the index finger. In addition, fat was removed from the abdomen and wrapped about the tendon of the index finger in the palm. This second procedure resulted in marked improvement in flexor power of the index and middle fingers, so that the patient is now able to bring them to within ½ inch of the palm. (Fig. 11.)

Case IV is that of a tendon graft of two fingers employing the extensor tendons of the toes.

CASE IV. W. O., a seventeen year old white grocery clerk, suffered a laceration of his right ring and little fingers in March, 1952, when it was accidentally cut while he was trimming produce. A diagnosis of lacerated flexor tendons to the ring and little fingers was made and primary repair done. The tissues healed without infection and the patient wore a posterior plaster of paris splint for four weeks.

When first seen in June, 1952, the tip of the ring and little fingers could be flexed only to within 1¾ inches of the distal flexion crease of the palm. (Fig. 12A.) Extension was normal. (Fig. 12B.)

Exploration revealed that although the flexor digitorum profundus tendons were intact there was severe scar tissue throughout the tendon sheath. It was therefore necessary to excise this tissue. Employing the extensors of the third and fourth toes, tendon grafts were done from the distal phalanx to the proximal portion of the flexor digitorum profundus in the palm. The flexor digitorum sublimi were intact.

At the end of three months the patient was able to bring his fingers to within ¾ inch of the distal flexion creases in the palm. He had increased strength and was using his fingers daily at work. (Figs. 12C and D.)

## SUMMARY

In a series of twenty-three cases with free tendon grafts in the fingers good functional results were obtained in all but four patients. Ten tendon grafts were performed in the thumb, with one failure. Secondary tenorrhaphies were performed on ten patients, with improvement being noted in all except one. The total range of motion obtained was less than that noted in tendon grafts.

The fundamental problems regarding flexor tendon grafts in the fingers and thumb have been stressed. Concepts developed during the past ten years have been elaborated upon and postoperative rehabilitation has been emphasized.

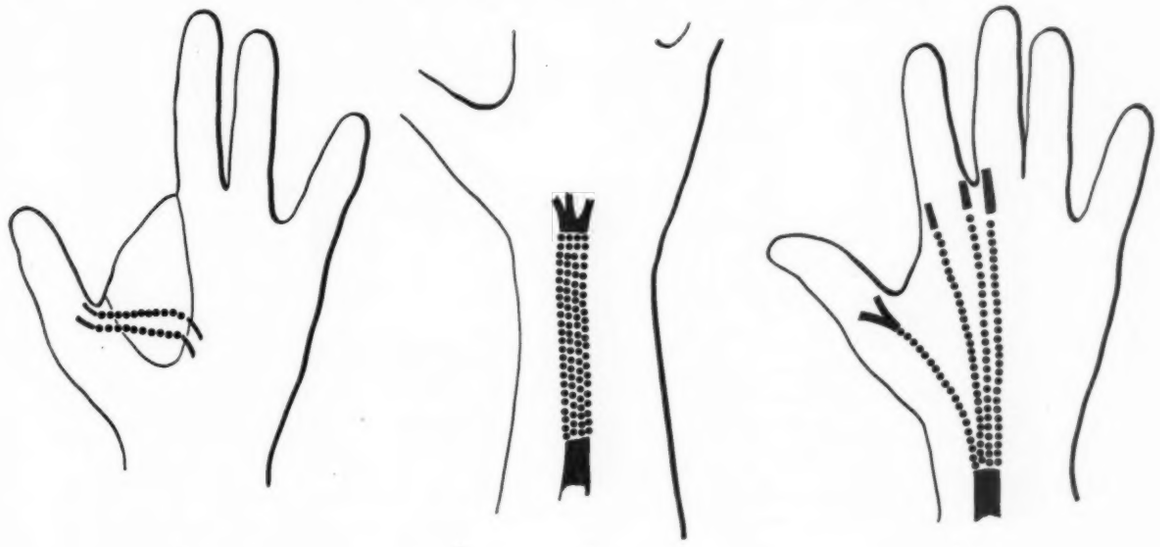
## REFERENCES

1. KOCH, S. L. Injuries of the parietes and extremities. *Surg., Gynec. & Obst.*, 76: 1–22, 189–196, 1945.
2. BUNNELL, S. Surgery of the Hand, 2nd ed., pp. 605–674. Philadelphia, 1948. J. B. Lippincott Co.
3. MASON, M. L. Principles of management of open wounds of the hand. *Am. J. Surg.*, 80: 767–771, 1950.
4. BUNNELL, S. The early treatment of hand injuries. *J. Bone & Joint Surg.*, 33-A: 807–811, 1951.
5. FLYNN, J. E. Flexor tendon grafts in the hand. *New England J. Med.*, 241: 807–812, 1949.
6. GRAHAM, W. C. Delayed tendon repairs. *Am. J. Surg.*, 80: 776–779, 1950.
7. LITTLER, J. W. Free tendon grafts in secondary flexor tendon repair. *Am. J. Surg.*, 74: 315–321, 1947.
8. GRAHAM, W. C. Flexor tendon grafts to the finger and thumb. *J. Bone & Joint Surg.*, 29: 553–559, 1947.
9. KOCH, S. L. Division of the flexor tendons within the digital sheath. *Surg., Gynec. & Obst.*, 78: 9–22, 1944.
10. BUNNELL, S. Surgery of the Hand, 2nd ed., pp. 412–438. Philadelphia, 1948. J. B. Lippincott Co.
11. KINMONTH, J. B. The cut flexor tendon; experiences with free grafts and steel wire fixation. *Brit. J. Surg.*, 35: 29–36, 1947.
12. ALLEN, H. S. Flexor tendon grafting to the hand. *Arch. Surg.*, 63: 362–369, 1951.
13. BOYES, J. H. Flexor tendon grafts in fingers and thumb; evaluation of end results. *J. Bone & Joint Surg.*, 32-A: 489–499, 1950.

# NERVE GRAFTS INTO HANDS

HENRY C. MARBLE, M.D. AND CHARLES B. BURBANK, M.D.
*Boston, Massachusetts*

WE believe that a free nerve graft is indicated in reconstructive hand surgery when (1) it is impossible by any other method to do either a primary or secondary suture and (2) when one wants a return of sensation to the hand. It is true that the sensation that returns is not normal but patients can tell the difference between hot and cold and therefore do not burn their hands; they know when they are touching objects, and so it converts a useless hand into a useful one. In our experience all the patients have been helped and none are worse. One or two have had an extraordinary return of feeling, even to the point of being able to tell small coins apart. We have not had any patient who has had return of motor power.



1 2 3

FIG. 1. Patient F. C. This man suffered an injury to his right hand in December, 1944. The index finger and the web between it and the thumb were completely avulsed and at the time of operation had to be discarded. The wound healed with the thumb flexed across the palm and adducted to the base of the third finger. In September, 1946, the scar between the thumb and the third finger was excised and a pedicle graft was applied to cover the defect. In May, 1947, two pieces of sural nerve were used to replace the lost segments of digital nerve to the thumb. The grafts are indicated by the dotted lines. Sensation began to return in September, 1947, and was complete to the tip of the thumb in 1948.

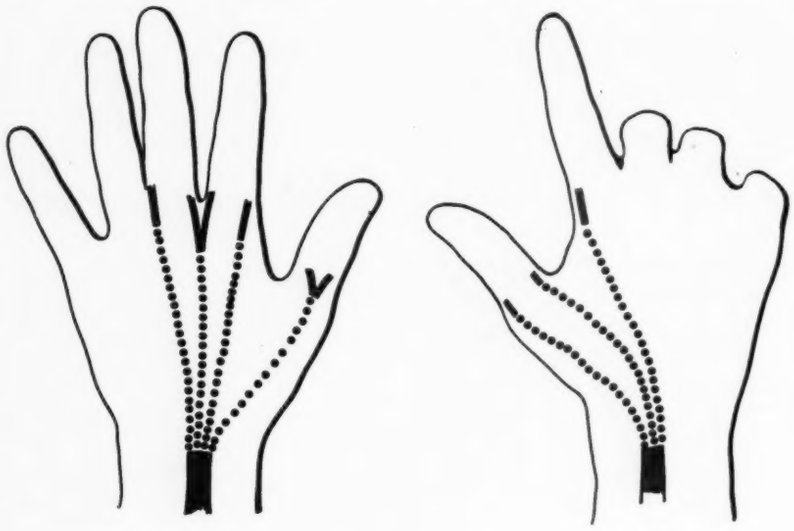
FIG. 2. Patient H. H. This man received a severe burn to his right forearm in October, 1944. The wound was first closed with a thin split-thickness graft and in 1945 this was replaced by a pedicle graft taken from the abdominal wall. The burn had destroyed some 4 inches of the median nerve and in December, 1946, the defect in the median nerve was repaired by using a four-strand cable graft from the sural nerve of the opposite leg. In June, 1946, the patient began to get a percussion response and by June, 1947, he had sensation to his fingertips.

FIG. 3. Patient D. C. In July, 1948, this man lost the palm of his hand when he caught it in a carding machine. Nerves, tendons and soft tissue were completely gone. The wound was first covered by a split-thickness Thiersch graft and this was replaced several months later by a pedicle graft taken from the abdominal wall. The digital nerves which had been lost at the time of the injury were replaced by strips of sural nerves in February, 1950. The dotted lines indicate the replacements. In June, 1950, sensation began to return and by February, 1951, sensation was complete to the fingertips.

A nerve graft may be done at the time of the original injury or as soon thereafter as the general condition of the patient and local wound warrants a second procedure. The time for nerve grafting is when the wound has

been properly surfaced and before other reconstructive procedures. This permits the patient to have sensation and thus helps as later operations are carried out.

In order to ensure success an autogenous nerve should be used, and theoretically it should be of the same caliber as the host nerve. This demand presents two problems: (1) to obtain a graft similar to the host it would be necessary to sacrifice the contralateral functioning nerve; this is out of the question; and (2) grafts large in diameter, even autogenous ones, do not do well. This is apparently due to the facts that their early nourishment is taken directly from the neighboring tissues and that this process will nourish only the outer layers of the large graft, for pathologically these failures show regeneration peripherally but necrosis and fibrosis centrally. This occurs in almost all nerves over 1 mm. in diameter. The answer then is to use sensory nerves of a diameter 1 mm. or less and to take it or them from areas where the loss of sensation will be least troublesome. By so doing one can use single strands for small nerves and multiple strands to make up the necessary diameter of larger host nerves. We have used four strands to bridge a median nerve defect 4 inches long and once a single strand 6 inches long for a digital nerve.



4 5

FIG. 4. Patient D. K. This man received a deep laceration across his palm in May, 1949. Apparently many nerves and tendons were injured and an attempt was made to suture all of these. The wound became grossly infected and in the course of several months all of the tendons and nerves to the thumb, index and middle fingers sloughed out. The wound eventually healed. In December, 1949, strips of sural nerve were used to replace the lost digital nerves as indicated by the dotted lines in the diagram. Sensation began to return in March, 1950, and by October, 1950, there was sensation to the fingertips.

FIG. 5. Patient M. H. In May, 1951, this man suffered an injury to his hand when he caught it in a burnishing machine. The whole palm and the third, fourth and fifth fingers were lost at the time of injury. The palm was resurfaced by a split-thickness graft and this was replaced by a pedicle graft two months later. In December, 1951, nerve grafts were made into the thumb and index finger using strips of sural nerve as indicated by the dotted lines in the diagram. Sensation began to return in May 1952, and when last seen in July, 1952, sensation was within 1 inch of the finger tips.

The largest and most easily accessible sensory nerve is the sural. This nerve supplies sensation to the lateral aspect of the calf, to the side and a small area on the sole of the foot. Loss of this sensation does not seem to trouble patients. Fourteen to 18 inches of nerve can be obtained through a long incision directly over its course. This can be cut up as needed. (Figs. 1 and 2.)

Homologous grafts do not work in humans though they may in animals. It is not clear

why. Large autogenous grafts do poorly in humans. Small autogenous grafts seem to do well as they derive their early nourishment directly from the tissues it is wise that each patient be carefully studied preoperatively for any physiologic deficit and that any such deficiency be corrected. The condition of the operative region is very important. The skin should be clean and evidence of any past infection should be gone. The patient should be told of the possibility of failure and more important be made to understand that only sensation will return and that this sensation will not be normal. (Figs. 3 to 5.)

Technically, the procedure must be meticulous. The two ends of the host nerve should be prepared first; they must be cut back until normal tissue is encountered. The longer the time since injury the more nerve will have to be sacrificed. Enough small strands should be used so that their combined diameter will equal that of the host. Very accurate end-to-end sutures must be done since the degree of success is in direct proportion to the accuracy of the union. Very fine silk or catgut may be used. Do not surround the anastomoses with foreign material as this interferes with the nutrition of the graft and causes scar tissue to form which interferes with regeneration. After care consists of immobilization for three weeks followed by whatever gentle active use is possible and gentle physiotherapy.

To prove the success of any grafting procedure novocaine injection of the proximal host nerve should abolish all sensation.

# MANAGEMENT OF SEVERE TRAUMA TO DISTAL DIGITAL EXTREMITIES

GEORGE F. CRIKELAIR, M.D., ROBERT T. CROWLEY, M.D.
*New York, New York*
AND HARRY MILLER, M.D.
*Allentown, Pennsylvania*

IN the medical literature of the past several years more and more attention has been directed to the treatment of the increasing number of injuries to the terminal phalanges of the fingers. Most of the individual articles deal with one specific method for the repair of a specified type of injury, the guillotine amputation. This is not the only type of injury to the terminal phalanx although it is a very troublesome one; nor is there any one method of repair applicable to all injuries.

To get a perspective on finger tip injuries, forty-four finger tip injuries in thirty-nine consecutive patients treated at the Columbia-Presbyterian Medical Center from January, 1950, to March, 1951, were reviewed. Even from this small number of injuries, it is apparent that finger tip trauma is of three general types: (1) door crush, (2) hand explosion and (3) guillotine type amputations. These will be discussed in order.

*Door Crush.* (Fig. 1A.) The door crush injury is most common in children. In this series there were nineteen such injuries, thirteen of which were in children. In addition to crushing of the soft tissues to a greater or less extent, there may be total or partial avulsion of the nail, irregular lacerations on either side of the finger extending toward the palmar surface, or almost total avulsion of the tip with fracture or crushing of the bone. Usually there is no significant loss of the substance of the tip. In treatment it generally suffices to cleanse the hand well, to débride minimally, to approximate gently the lacerated or partially avulsed tip and to apply a light pressure dressing. Depending on the severity of the injury there may be nail deformity and perhaps a slight "trap door" elevation of the tip.

*Hand Explosion.* (Fig. 1B.) Irregular and extensive loss of soft tissue from the tip and occasionally from more of the finger occurs in hand explosions or in injuries in which the tip is tightly wedged and then forcibly withdrawn. The bone is usually intact but exposed. This was the least common terminal phalanx injury in this series. There were two cases. One boy injured three fingers when a homemade bomb exploded in his hand and the other patient injured the finger by wedging. There are two methods of repair for such an injury. Either amputation of the bone and a primary amputation closure or the method elected in these two patients, a pedicle flap to supply both fat and skin. Despite the fact that pigmentation and slow return of sensation may follow so that two operative procedures with a subsequent revision are necessary, in this type of finger tip injury an abdominal or chest pedicle flap has its strongest indication.

*Guillotine Type Amputations.* (Fig. 1C.) Guillotine amputations of the terminal phalanx vary in extent from skin loss to loss of skin and pulp, a portion or all of the nail with exposure or partial removal of the terminal bony phalanx. There are many methods reported and used for repairing this defect. Eighteen patients in this series had this type of injury, some multiple, so that there were twenty-three finger tips treated. Seven different methods of treatment, none original, were employed in these cases. Four other methods of treatment found in the literature were not used and will be discussed first.

1. In 1919 Staige Davis advocated wrapping the finger tightly with celluloid to confine the granulations to the desired size and direction. This has been revived recently by Lace using adhesive tape. It would seem that there are enough other methods for getting prompt coverage with good results so that the use of a procedure which invariably results in a painful, septic wound seems unnecessary.

2. Composite ear lobe grafts reported by

Clarkson have not been used. His description of delayed healing with a resulting soft stump without sensation is sufficient warning against this method.

3. Kutler's V-Y advancement of the skin and fat along the sides of the finger has not been used, because of the fear of further loss of one of these pieces and the added incisions and scarring which may result. He has reported good results with the method, however, and it warrants trial in some cases.

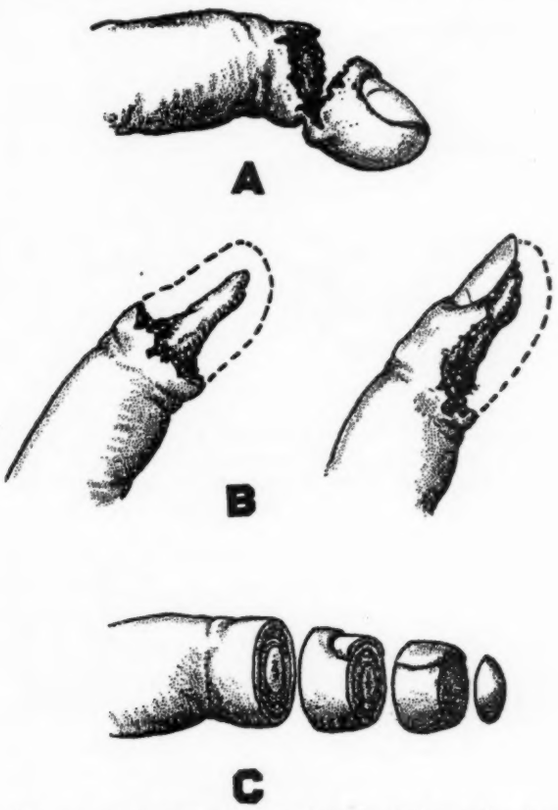



FIG. 1. A, door crush type injury of finger tip; B, hand explosion or injury by having the tip tightly wedged and forcibly withdrawn; C, guillotine type amputation; the degrees of severity are diagrammatically shown.

4. Replacement of the tip has not been done in any of these injuries because the patient was never thoughtful enough to bring it with him. This replacement r pair will be discussed later.

## METHODS USED

*No Surgery.* In instances in which there was small or superficial skin loss in adults or in very small infants with any loss, large or small, treatment with a vaseline or xeroform dressing has sufficed. In children re-epithelization is rapid with minimal scarring and this balanced against a general anesthetic and difficult post-operative dressings makes this a valuable form of therapy.

FIG. 2. Volar tip flap; the bone is shortened, palmar surface undermined, advanced and sutured to the nail.

*Volar Finger Flaps.* In people who do heavy manual labor in which a tough finger end which heals rapidly is more important than functional or cosmetic length, removing a bit more of the bone and advancing the palmar flap over the end to suture to the nail or dorsal skin has given good results. (Fig. 2.)

*Split Thickness Skin Grafts.* This method of repair has often been rejected because of the sensitive, scarred and sometimes painful tip which results. However, since it is easily obtained, takes well and will contract to a great extent, it is sometimes of value. If the remaining piece of nail is small, the graft will pull the nail down and the graft will be over the tip of the finger as in Figure 3B. However, if there is enough of the nail present (Fig. 3A), as the graft contracts it will pull the palmar portion of the tip upward and the graft then disappears beneath the nail. Slight undermining of the palmar surface helps advance soft tissue toward the nail.

*Full Thickness Grafts.* This method has attracted the most attention. It, too, is easy to obtain; and since the entire dermis is included, it is more satisfactory than a split graft. Full thickness grafts require more exact handling as regards the bed for the graft, the tension and the pressure applied over the graft. The healing time is longer than for some of the other methods and there is often loss of the very center if placed directly over the bony phalanx.

*Cross Finger Pedicles.* If the adjacent finger has not been damaged and if the defect to be filled is not too large, a retrograde flap gives excellent coverage especially over exposed bone, since there is fat padding and skin similar to that of the finger tip being used for replacement. That two operative procedures are required and that a split skin graft must be used to cover

the good finger in no way detracts from its value. (Fig. 4.)

*Palm Flaps.* This method is comparable to the cross finger method in that two procedures are needed to supply fat and skin similar to the finger tip. There is a scar or graft present in the palm which is usually not troublesome. It is not a good method to use in an elderly person or someone with arthritis because of the flexed position of the finger which must be maintained for about three weeks while the flap is attaching itself to the finger tip. (Fig. 5.)

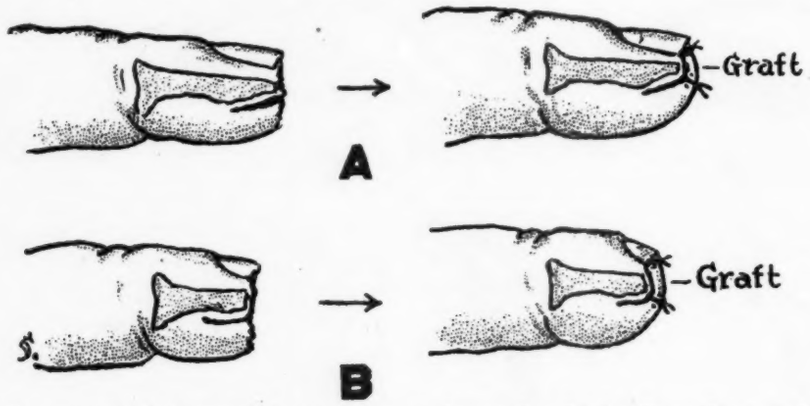



FIG. 3. Split thickness graft: A, palmar surface undermined, split graft sutured to this edge, tensed and then sutured to nail; palmar flap pulled toward the nail as graft contracts. B, short piece of nail remaining is pulled downward by graft contracture.

### REAPPOSITION AND FIXATION OF THE DISTAL SEVERED PART

In certain cases in which the injury is seen very soon after incidence, the severence cleanly made, the severed part available, minimally damaged, and not too great in extent, an attempt may be made to restore it as a whole to the proximal extremity stump. Whether this procedure is to be tried in preference to some other method of covering the extremity stump is a matter of individual judgment and preference. If, however, it is decided from estimation of the injury that an attempt is warranted to restore the distal severed part as a whole to the extremity stump, the method by which the opposition and fixation of the parts is to be accomplished requires close consideration.

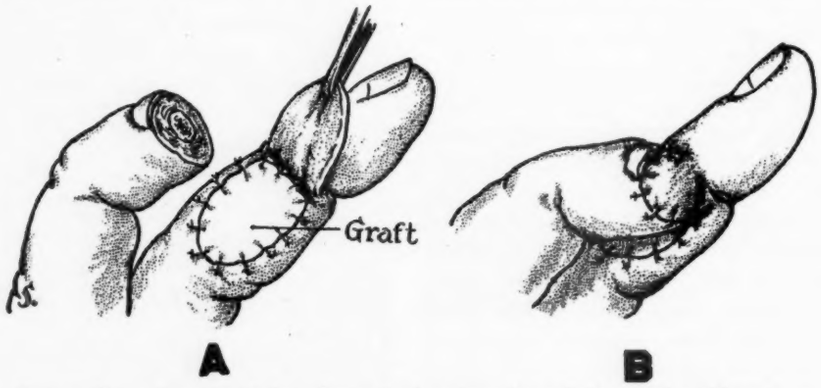



FIG. 4. Cross finger pedicle, schematic drawing: A, pedicle raised and base covered with split thickness skin graft; B, flap sutured to amputated stump.

*Bi-pedicle Flap.* This was used on one finger in this group. There was almost complete loss of the terminal phalanx with a transverse laceration about 1.5 cm. proximally on the dorsum. It was most expedient to undermine this flap, advance it over the tip and put a split skin graft into the defect on the dorsum of the finger. (Fig. 6.)

Any particular procedure chosen for this specific purpose must, if there is to be any prospect of success, observe the following

essentials: (1) The severed portion must be opposed to the extremity stump in as near an anatomic alignment of bony and soft tissue as possible. (2) The fixation of the anatomic alignment of severed part and extremity stump must be such that there is a firm even pressure

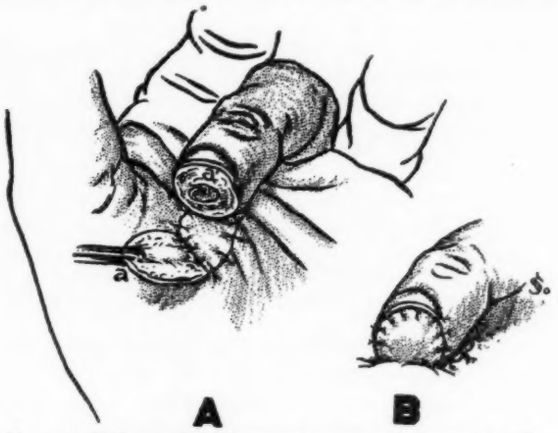



FIG. 5. Palmar flap from thenar eminence: A, flap raised and palmar defect grafted; B, pedicle in place on amputated finger stump.

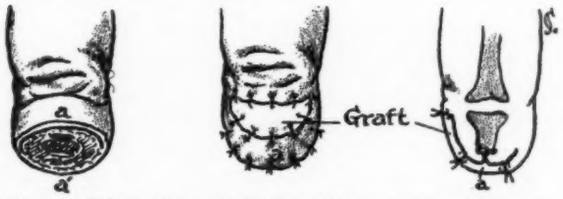



FIG. 6. Dorsal bi-pedicle flap. Flap *a* undermined but attached medially and laterally and advanced over tip to *a'*; split thickness graft in dorsal defect.

and no slipping between their opposed surfaces for any prolonged period of time. (3) There must be minimal interference with the circulation at the line of opposition of severed part and extremity stump. (4) There must be minimal trauma to the extremity stump and severed part during the process of opposition and fixation.

It is the purpose of the subsequent description to present a simple, rapidly accomplished technic, applicable in the type of finger trauma specified which observes these essentials. In the experience of the writers its use in properly selected cases has been attended by very satisfactory results in ten of fourteen cases.

Under adequate anesthesia and with the injured digit, hand and forearm properly prepared and draped, the severed surfaces of the amputated part and extremity stump are carefully débrided with fine plastic instruments conserving as much of the tissue of each as possible. When any excessive hemorrhage from the extremity stump has been controlled so that its surface is relatively dry or presents a uniform slowly seeping surface, the severed surface of the amputated portion is coapted to it in as close an anatomic alignment as possible.

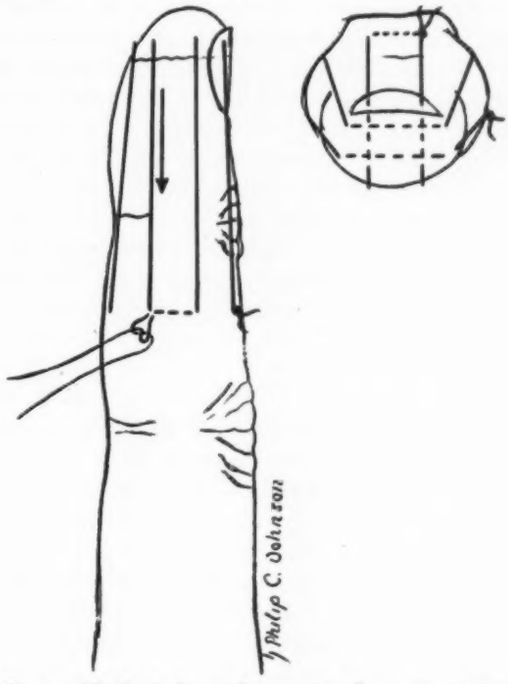



FIG. 7. Technic in replacement of an amputated tip.

To fix the coapted parts in this position No. 00 silk or a small fine curve cutting needle is employed for the stay sutures. (Fig. 7.) The first of these is passed through the skin and tough fibrous tissue on the dorsum of the finger at a point about one-half the distance between the first and second interphalangeal joint. The suture is then carried distal to the opposed severed part where it is passed through the tissue of its tip. From here the suture is carried downward on the volar surface of the finger to a point corresponding to that on the dorsal surface where it is similarly passed through the tissue and again brought up to the tip of the opposed severed portion. As before, it is passed through the tissue of this part and brought downward on the dorsum of the finger where it can be tied to the free end. The second or later stay suture is placed in a like manner beginning on the lateral aspect of the finger at a point about midway between the first and second interphalangeal joints, passing distal to affix the tip of the severed portion downward

to the other lateral aspect of the finger, distal from there to transfix the tip and from there to the side of the finger to join the free lying end, with the severed segment firmly opposed to the extremity stump. The stay sutures, anteroposterior and lateral, are then tied thus fixing the opposed surfaces firmly in position. When the alignment and fixation are satisfactorily accomplished by the stay sutures, the circumferences of the finger stump, the affixed severed portion and their line of juncture are divided into four approximately equal segments. In each of these segments a fine suture of No. 0000 is passed through the line of approximation of the severed portion and extremity stump and tied. A dry dressing is applied and the part properly splinted in a position of hemiflexion.


## SUMMARY

There are several types of injuries, lacerations and amputations of the terminal phalanx. The goal in repairing such an injury is a serviceable, non-tender, durable tip as near normal length as possible in as short a time as possible. No one type of repair will suffice for each injury. There are many excellent methods of repair which can be used, and the choice must balance the type of injury, the age and the occupation of the patient, the knowledge of the surgeon and his familiarity with these many procedures to get the best result in each injured terminal phalanx. Whatever method of repair is used, postoperative rest, immobilization and elevation are essential to good healing.


## REFERENCES


1. Stenberg, G. Full thickness grafts in finger tip injuries. *Acta chir. Scandinav.*, 99: 435–444, 1950.
2. Clarkson, P. Composite ear grafts in the repair of traumatic amputations of the finger tips. *Guy's Hosp. Gaz.*, 63: 266–268, 1949.
3. Gurdin, M. and Pangman, W. J. The repair of surface defects of fingers by transdigital flaps. *Plast. & Reconstruct. Surg.*, 5: 368–371, 1950.
4. Lace, W. T. Guillotine amputation of the finger tips: an improved method of treatment. *Am. J. Surg.*, 81: 348–350, 1951.
5. Kutler, W. A method for the repair of finger amputation. *Ohio State M. J.*, 40: 126, 1944.
6. Kutler, W. A new method for finger tip amputation. *J. A. M. A.*, 133: 29–30, 1947.
7. Powell, F. I. A "minor" injury of the terminal phalanx. *Brit. M. J.*, 1: 618, 1949.
8. Auchincloss, H. Surgery of the Hand. Nelson's Loose-Leaf Surgery, pp. 459–471. New York, 1927. Thos. Nelson & Sons.
9. McCarroll, H. R. Immediate application of free full-thickness skin graft for traumatic amputation of the finger. *J. Bone & Joint Surg.*, 26: 489–494, 1944.
10. Reed, J. V. and Harcourt, A. K. Immediate full thickness grafts to finger tips. *Surg., Gynec. & Obst.*, 68: 925, 1939.
11. Cole, P. P. Experiences in reparative surgery of the upper limb. *Brit. J. Surg.*, 28: 595, 1940.
12. Jones, R. A. A method for closing a traumatic defect of a finger tip. *Am. J. Surg.*, 55: 326–336, 1942.
13. Koch, S. L. The immediate treatment of injuries of the hand. *Surg., Gynec. & Obst.*, 52: 598, 1931.
14. Gilcrest, E. L. Plastic operation for repair of a traumatic amputation of end of finger. *Surg. Clin. North America*, 6: 539–554, 1926.
15. Zadik, F. R. Immediate skin grafting for traumatic amputation of finger tips. *Lancet*, 1: 335–336, 1943.
16. Lauten, W. F. Finger tip reconstruction (a new operation). *Indust. Med.*, 8: 99–100, 1939.
17. Koch, Sumner L. The Covering of Raw Surfaces with Particular Reference to the Hand. *Surg., Gynec. & Obst.*, 43: 677–686, 1926.
18. O'Malley, T. S. Full thickness skin grafts in finger amputations. *Wisconsin M. J.*, 33: 337, 1934.
19. Davis, J. S. Plastic Surgery: Its Principles and Practice, pp. 664–670. Philadelphia, 1919. P. Blakiston's Son & Co.
20. Bunnell, S. Surgery of the Hand. Philadelphia, 1944. J. B. Lippincott Co.
21. Tempest, M. N. Cross finger flaps in the treatment of injuries to the finger tip. *Plast. & Reconstruct. Surg.*, 9: 205–222, 1952.
22. Auchincloss, H. Surgery of the Hand. Nelson's Loose Leaf Surgery, pp. 486–487. New York, 1927. Thomas Nelson & Sons.

# EPIDERMOID (EPITHELIAL) CYST OF THE HAND SKELETON

ROBERT E. CARROLL, M.D.
*New York, New York*

FOR over seventy-five years the presence of epidermoid cysts in the soft tissues of the hand has been an accepted entity. These discrete cysts have been found on the volar or grasping surface of the palm and fingers. Traumatic implantation of epidermal tissue is described as the etiologic agent. They are not a rare occurrence. In industrial areas when frequent handling of material is necessary the occurrence tends to increase.

This type of implantation cyst, the epidermoid or epithelial cyst, also is found to exist within the substance of the phalanx of the digit. It is not often observed. Rarely does one physician or even a hospital have the opportunity of treating a patient with such a condition. This may be the result of failure to recognize this tumor. Where the information can be found, no correct preoperative diagnosis has been made by roentgenologist or surgeon. It therefore seemed profitable that the subject receive amplification. The purpose of this article is to provide the basis for diagnosis and treatment of the epidermoid cyst within bone as it occurs in the hand. It does not propose to include defects of the hand skeleton caused by extrinsic pressure of epithelial cysts in soft tissue.

It is generally accepted that Sonntag[17] first described this entity in 1923. A young female received a crushing blow injuring the third digit of the left hand. Amputation of the terminal phalanx and the distal half of the middle phalanx was necessary. For twenty-four years the stump had been useful and painless. A swelling developed in the remaining half of the middle phalanx, which was thought to be a sarcoma. Upon removal it was found to be an epidermoid cyst in the remaining half of the middle phalanx.

Of the remaining eighteen cases all were found within the terminal phalanx. Burrows[4] reported a case in 1926 having the greatest lapse of time from initial trauma to surgery. The patient stated that thirty-six years prior to admission he had pierced his left thumb with a wire. In 1930 Friedländer[8] reported the youngest patient on record, a boy of thirteen. Both Hammann[10] and Harris[11] each reported a case in that same year. In 1931 Behrens[2] and Siebner[16] each added a new case. Behrens also described the case previously reported by Friedländer. In 1933 Curtis and Owens[6] added a case which required a second excision to control the defect. Couch[5] described a thumb case, from a previous hammer blow, in 1934. Bissell and Brunschwig[3] added two more cases in 1937. Reich[14] in 1938 was able to add a new case. The year 1940 was productive in one case from Becker[1] and two cases from Frostberg.[9] Single cases were provided by Yachnin and Summerill in 1941,[18] by Ribeiro[15] in 1942, by Pohlmann and Wachstein[13] in 1944, and by Duperrat and Gandrille[7] in 1948. This would give a total of nineteen individual cases that have been described in the literature.

For a more complete picture of the problem presented by this tumor, six new cases are described. They are quite similar to all cases previously reported in the literature.

## CASE REPORTS

CASE 1. J. M., a thirty-five year old delivery man, crushed the terminal phalanx of the fifth digit, right hand, between two cases of bottles. There was some bleeding around the nail but he was able to continue work. About the third week the nail itself had worked loose and fallen off. A new nail grew in its place. Toward the end of the twentieth month the terminal phalanx was found to be noticeably enlarged. No pain was present. The presence of an enlarged phalanx caused this patient to seek medical attention.

Two years following injury examination of the terminal phalanx of the fifth digit, right hand, was found to be enlarged. There was full range of motion in the distal interphalangeal joint. Some tenderness was elicited on pressure to the phalanx. The skin was not discolored.

Roentgenograms (Fig. 1) revealed an expansile lesion in the terminal phalanx which caused the cortex to be thin and bulging. A break in the cortical continuity could be found on the volar aspect. The lesion was diagnosed as enchondroma.

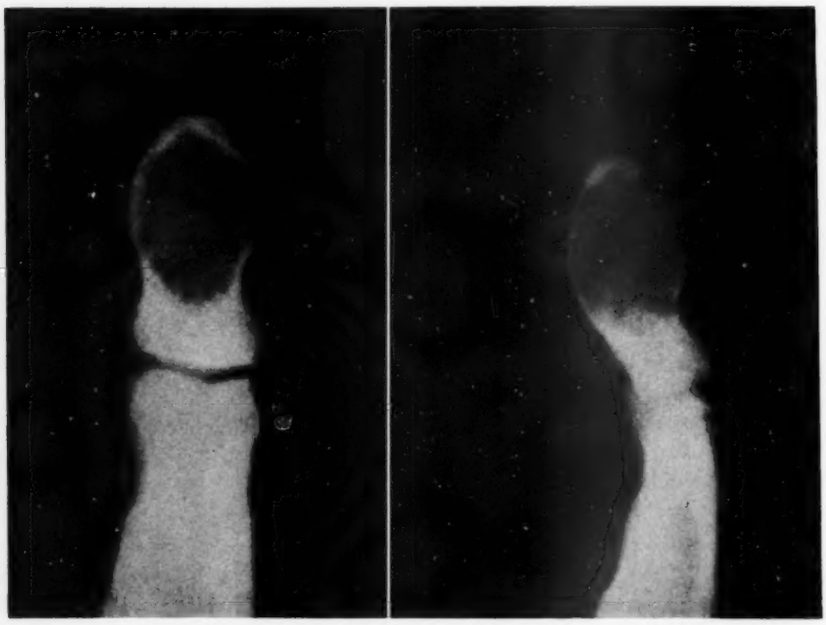


FIG. 1. Anteroposterior and lateral roentgenograms of the terminal phalanx, fifth digit. A cystic lesion is found to expand the cortex. The break in the wall is a fracture line caused by minimal pressure.

Amputation was performed through the distal quarter of the middle phalanx. The patient returned to work in three weeks.

Section of the terminal phalanx (Fig. 2) revealed white caseous material in the center of the cavity. The wall was formed by a tough membrane. The cortex of the phalanx surrounded the membrane even though paper thin in places. Upon review under a microscope the membrane was found to consist of stratified epidermal epithelium. (Fig. 3.)

CASE II. A. B., a twenty-four year old female, caught her right thumb in a closing drawer when sixteen years of age. The nail became discolored and dropped off. Swelling of the phalanx was noted two years after injury. Pain had been noticed six months prior to admission.

Upon admission the terminal phalanx of the right thumb was markedly swollen. Range of motion in the adjacent joint was normal. Pressure to the area caused pain.

Roentgenograms showed that the distal two-thirds of the terminal phalanx were replaced by an expansile lytic lesion. A thin rim of cortex could be seen which was called a bone cyst.

Amputation of the terminal phalanx was performed. The lesion contained caseous material within a tough membrane. On microscopic section the membrane was found to consist of stratified epithelium.

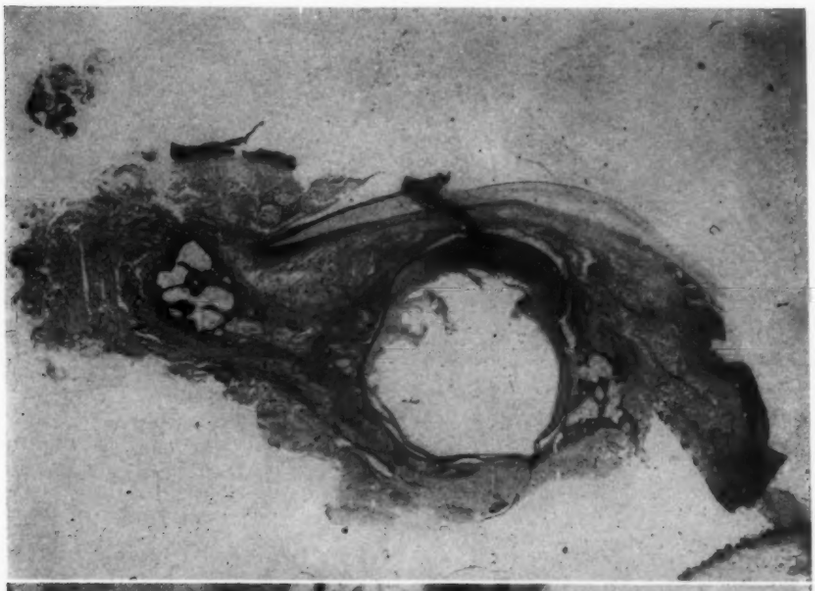
CASE III. (Courtesy of Dr. D. J. Greiner.) D. H., a thirty-two year old mechanic, could not remember specific previous trauma to the third digit of his right hand. While pulling on a lug wrench he felt sudden pain in his terminal phalanx As it continued to ache, he sought medical advice.

Upon admission the terminal phalanx was enlarged and tender. Roentgenograms revealed a lytic lesion of the terminal phalanx, which had a fracture line across the two thinned walls. The diagnosis was fracture through an enchondroma. The cyst was curetted. The pathologic report was that of an epidermal cyst of inclusion type. No follow-up history is available.

CASE IV. (Courtesy of Dr. J. P. Webster.) R. T., a forty-five year old man, remembered crushing the terminal phalanx of the third digit, left hand, at the age of twenty while playing baseball. About two years after that he remembered injuring the same digit by a hammer blow. The phalanx had become swollen and remained about the same for many years. Because he thought the swelling to be increasing and a slight pain was noticed on pressure, the patient sought medical advice.

Admission examination of the terminal phalanx, third digit of the left hand, found it to be swollen, tender and red. The range was normal in the adjacent joints. Roentgenograms demonstrated an expansile lesion involving most of the terminal phalanx. The diagnosis was enchondroma or possibly a malignant change. Amputation of the entire ray at the metacarpal base was performed. The lesion was found to be an epithelial cyst within the phalanx.

CASE V. (Courtesy of Drs. R. H. Ramsey and F. C. Reynolds.) J. B., a twenty-seven year old



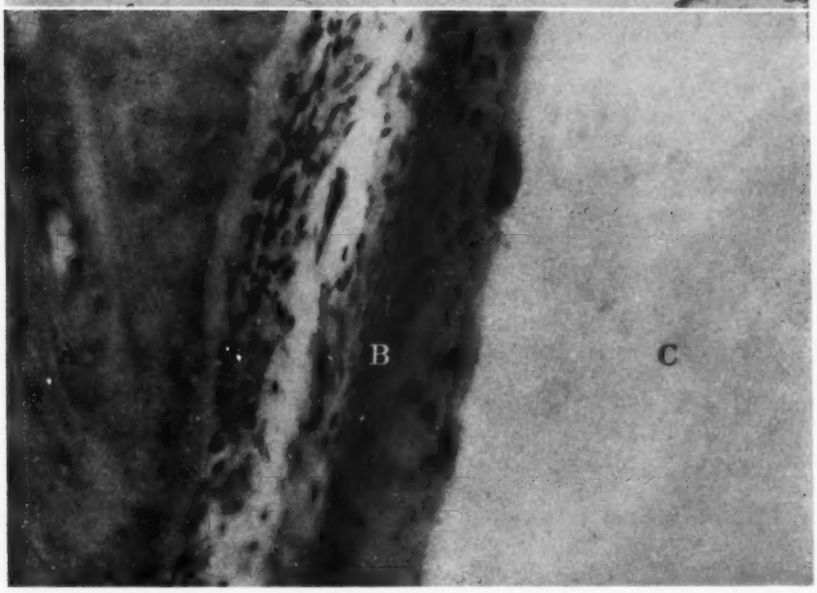



FIG. 2. Saggital section of the terminal phalanx demonstrates the position of the cyst within the phalanx. The tough membranous lining can be seen. The central caseous material fills the cyst; (very low magnification).

FIG. 3. Section of the membrane lining the cyst. A, normal bone of the phalanx; B, the stratified epithelium forming the membrane; C, the central caseous material; (low magnification).

man, stated that an enchondroma had been removed from the terminal phalanx on the left fifth digit when he was seventeen. This material could not be obtained for review. Full painless use of the hand had been possible up to six months prior to admission. At that time the patient had become aware of painless enlargement of the distal phalanx.

Admission examination found the terminal phalanx of the left fifth digit to be enlarged and slightly tender to palpation. Roentgenograms were considered to reveal recurrent enchondroma. At operation a cystic cavity was found in the terminal phalanx. This was filled with white sebaceous material. The cavity was thoroughly curetted and the skin closed. No follow-up information is available.

CASE VI. (Courtesy of Dr. F. E. Stinchfield.) D. A., a thirty-seven year old man, sustained a crushing blow to his left fifth digit when he was about seventeen years of age. The terminal phalanx had always remained enlarged in this digit. Six weeks prior to admission the distal

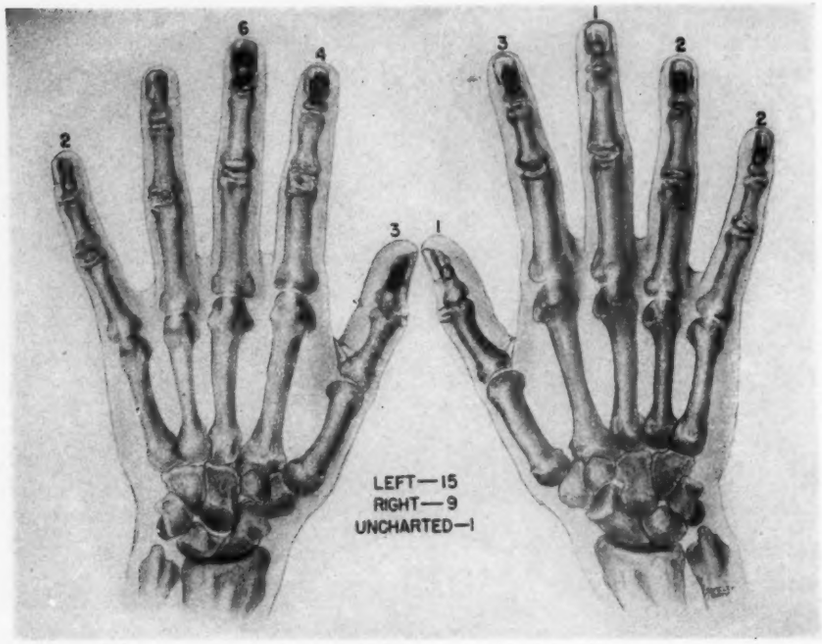



FIG. 4. Diagram of the location in the digits for the nineteen cases from the literature and the six new cases. In one case from the literature the digit was not identified but the cyst was in the terminal phalanx. The circles represent newly reported cases. The solid dots represent the previously reported cases from the literature.

phalanx had become tender to touch and ached with use. As the roentgenograms revealed a destructive lesion in the terminal phalanx, his family physician had given him a full course of penicillin. Pain subsided but the phalanx remained swollen.

Upon admission to the hospital the terminal phalanx of the left fifth digit was found to be enlarged and reddened. Full painless motion of the adjacent joint was present. Roentgenograms suggested osteomyelitis or enchondroma.

At operation the distal phalanx was found to contain white, cheese-like material. A heavy capsule was removed from the cavity in the phalanx by curettage. Pathologic examination found stratified squamous epithelium forming the capsule. The findings indicated an epithelial cyst. Roentgenograms taken eighteen months following surgery indicate the presence of a cystic defect but no symptoms are present.

## COMMENTS

From the material assembled, the complete clinical and pathologic entity of the epithelial or epidermoid cyst can be appreciated. Of the twenty-five patients, sixteen were men and nine were women. The youngest patient was thirteen and the oldest fifty; the average age was about thirty-four. The left hand was involved more than the right hand. The terminal phalanx of the left third digit was found to be the most common site. (Fig. 4.)

*History.* The majority of patients remembered specific trauma involving the affected digit. The degree of trauma varied from a severe crushing blow to a rather slight injury. It is surprising that the majority occurred as a result of incidents involving a hammer blow, closing door, orange squeezer, fan blade, thrown baseball or sliding case of bottles. Because of the rather common occurrence of these traumas, those patients giving a negative history for previous trauma may not have remembered the incident. Following injury the average time for swelling of the phalanx to be noted was about eight and a half years. The earliest period consisted of two months while the longest interval reached thirty-five years. In a few cases the affected digit sustained several episodes of trauma. The patients waited on the average of about ten years after trauma before the need for surgery occurred.

*Symptoms.* Pain and increased size of the terminal phalanx caused the patient to seek advice. These symptoms often followed a minor episode of grasping. The pressure was enough to fracture an already thin cortex. The pain was mild enough to allow the patient to wait

several months before seeking advice. It did not limit the use of the hand.

*Clinical Findings.* In all cases the terminal phalanx of the digit was involved. All were noticeably enlarged. A little more than half were tender to palpation or with touch. There were a few that appeared erythematous to the examiner. In all cases the adjacent joint was not involved and a full range of painless motion could be obtained.

*Roentgenographic Findings.* The lesion is a lytic area in the terminal phalanx. It is usually round and occurs in the area of the tuft or mid-shaft of the phalanx. It may replace all but the base of the phalanx on occasion. The cortex is expanded and thinned but there is no evidence of periosteal reaction. A fracture line may often be found in the thinned area of cortex. Although the position of the cyst may be eccentrically placed, there is no doubt that the lesion exists within the cortex. There should be no problem of confusing the lesion with a lytic area caused by extrinsic soft part tumor.

*Diagnosis.* Although the history and clinical examination are of the greatest value, the diagnosis is mainly derived from the roentgenographic findings. From data obtained to date no correct preoperative diagnosis has been made of this specific entity. Most commonly it has been called an enchondroma. This is a sound observation as the enchondroma is the most frequent tumor causing a centrally located defect in the osseous skeleton of the hand. The lesion has also been called a bone cyst. Jaffe and Lichtenstein[12] doubt the existence of a simple bone cyst in the hand. The author also believes that a simple bone cyst in the hand has yet to be recorded.

Giant cell tumor in the hand rarely simulates the radiographic entity seen in long bones of the skeleton. It usually has a gross expansile nature that often acquires the modifying adjective, "aneurysmal." There is a class of lytic defects called osteitis fibrosa. These are localized findings where the bone cavity contains fibroblastic variants. Usually they form a trabeculated pattern on the roentgenograph.

It is surprising to find the defect called osteomyelitis. Certainly there is no sign of periosteal reaction that would accompany an inflammatory action. Even the possibility of Brodie's abscess would show some bone reaction.

The single lytic lesion in the terminal phalanx should not be confused with primary tumors of bone. Both primary and metastatic tumors would give evidence of progressive destruction. Primary tumors of bone are extremely rare in the hand. The metastatic single focus always shows a ragged margin where invasion is active. The outline of the epithelial cyst is smooth and well demarcated.

*Treatment.* The most definitive treatment consists of amputation through the distal portion of the next most proximal bone. If the cyst is small, there is a good chance that curettage would be of value. Curettage may be given as the primary method of treatment if there is to be an adequate period of after care. The patient should be informed of the possibility of a phalangeal amputation.

*Operative Findings.* A thin shell of cortex is usually present. This is easily opened. The capsule of the cyst itself is quite easily identified; it is a tough, firm membrane. The material contained within the cyst is best described as white, caseous material. It is softer than the tapioca-like material found in the enchondroma. These findings in themselves make the correct diagnosis. The operative finding is the gross pathology of this case.

*Microscopic Pathology.* The wall of the cyst consists of stratified squamous cell epithelium. Sweat glands or hair follicles are never found. The major portion of the wall is stratum granulosum with stratified cells of a stratum corneum. The cyst contains keratinized debris. Diagnosis by microscopic section should give little difficulty.


## SUMMARY

The problem of epidermoid (epithelial) cyst in the phalanx of the hand has been reviewed. Both nineteen recorded cases and six new cases have been used to discuss the diagnosis, treatment and pathologic findings of this entity.


## REFERENCES


1. Becker, F. Cyst of pavement epithelium of bones of fingers. *Chirurg.*, 12: 275–279, 1940.
2. Behrens, A. Traumatic epithelial (dermoid) cysts. *Virchows Arch. f. path. Anat.*, 280: 145–151, 1931.
3. Bissell, A. D. and Brunschwig, A. Squamous epithelial bone cysts of the terminal phalanx. *J. A. M. A.*, 108: 1702–1704, 1937.
4. Burrows, H. Implantation dermoid of the terminal phalanx of the thumb. *Brit. J. Surg.*, 13: 761–762, 1926.
5. Couch, J. H. Epidermoid cyst in bone. *Canad. M. A. J.*, 30: 516–518, 1934.
6. Curtis, F. E. and Owens, C. I. Sebaceous cyst of

the distal phalanx. *J. Bone & Joint Surg.*, 15: 998–1000, 1933.
7. Duperrat and Gandrille. Les kystes épithéliaux intra-osseux de la phalangette. *Semaine d. hôp. Paris*, 24: 78–79, 1948.
8. Friedländer, C. Traumatic epithelial cyst in terminal phalanx of forefinger. *Zentralbl. f. Chir.*, 57: 209–212, 1930.
9. Frostberg, N. Bone defects in terminal phalanges. *Acta Radiol.*, 21: 126–142, 1940.
10. Hammann, Traumatic epithelial cysts of phalanges of hand. *Deutsche Ztschr. f. Chir.*, 223: 308–317, 1930.
11. Harris, R. I. Sebaceous cyst of terminal phalanx of thumb. *J. Bone & Joint Surg.*, 12: 647–648, 1930.
12. Jaffe, H. L. and Lichtenstein, L. Solitary benign enchondroma of bone. *Arch. Surg.*, 46: 480–493, 1943.
13. Pohlmann, H. F. and Wachstein, M. Epidermoid (squamous epithelial) bone cyst of phalanx. *Ann. Surg.*, 119: 148–154, 1944.
14. Reich, W. W. Squamous epithelial bone cyst of the terminal phalanx. *California & West. Med.*, 48: 120, 1938.
15. Ribeiro, E. B. Epidermoid cyst at the third phalanx. *An. paulist. de med. e cir.*, 44: 209–224, 1942.
16. Siebner, M. Traumatic epithelial cysts of finger bones. *Chirurg.*, 3: 810–814, 1931.
17. Sonntag. Epithelial cyst in finger stump. *München. med. Wchnschr.*, 70: 1055–1056, 1923.
18. Yachnin, S. C. and Summerill, F. Traumatic implantation of epithelial cyst in a phalanx. *J. A. M. A.*, 116: 1215–1218, 1941.

## DISCUSSION OF PAPERS BY DRS. POSCH, MARBLE AND BURBANK, CRIKELAIR, CROWLEY AND MILLER, AND CARROLL

Henry C. Marble (closing): I want to call particular attention to the first case that Dr. Burbank noted. This patient's thumb was pulled into the palm of the hand and he had a grievous scar between the thumb and the tip of the mid-finger. He had a useless hand because when he tried to close his fingers he closed down against the thumb. This had to be released.

A full-thickness graft was put in the web. He had a thumb out in abduction but it was asensitive. The asensitive scar, the asensitive thumb, might be burned, cut or be the source of atrophic ulcers. For these reasons we ran these three tiny grafts across and were very happy with the result. The man had sensation in his thumb, power of opposition and could return to work. We have found this a useful method, particularly for small lost nerves in the hand.

I was very much interested in Dr. Posch's paper because I have been to Detroit and seen his cases. His was an adequate description of how to handle these lost tendon cases.

Dr. Crikelair has advocated putting the finger underneath the leg or underneath the breast to get a pedicle graft. My experience with that has been very unfortunate. Often I have had to take off those grafts and put on something new. At the time of the accident the surgeon has little time and opportunity to defat the pad adequately which he wants to put on the end. I have long since discontinued that process. I believe that in a traumatic injury to the hand it is better to resurface the hand with a split graft and not try to prepare the pedicle. Any pedicle procedure which I have done, and most of those I have seen done by my fellow surgeons, have not been very successful. I now ask my associates and residents at the hospital simply to débride the wound and resurface it. It is a great, great temptation to try to go the whole hog, so to speak, but you can promise yourself that 95 per cent of them will fail.

Dr. Carroll's paper intrigues me because when I saw his first x-rays I said, "He has a glomus tumor." I have never seen one such as these. I have seen many epidermoid cysts under the skin but I have never seen one in the bone, yet Dr. Carroll found six in a couple of months.

Michael L. Mason (Chicago, Ill.): I think these four papers have been very interesting. The first paper dealing with tendon repairs in the fingers presents several points worth mentioning. First, the question of secondary repair: When is it done? It should be done as soon as possible; in some instances it may be accomplished within three or four weeks after the injury, that is, as soon as the tissues are nicely healed and soft. If the tissues fail to soften or if they take a long time to do so, secondary repairs are likely to be less successful. We have found that frequently the profundus tendon, hen it is divided close to the distal phalanx, can be repaired either primarily or secondarily by suture. It is not always necessary to insert a tendon graft. When the division has been within the proximal three-quarters of the fingers, as Dr. Posch has said, one must have a tendon graft. We have come to a very simple technic for attaching the graft to the distal phalanx. There are many methods that have been advocated for attaching the stump to the distal phalanx: drilling holes through the bone, pulling the tendon around the bone, and so forth. About three years ago Dr. Cyril Hauser studied experimentally the problem of attachment of tendon to bone. He found that the simplest method by which tendon could be attached was the best method. He simply sutured it to the periosteum with four very fine nerve silk sutures, and within three weeks the attachment of the tendon became exceedingly strong. I think Dr. Posch also hit upon the real problem in tendon repair, that of tension at which tendon grafts should be inserted. I think the man who solves that problem should have a gold medal because I am sure that I am not entirely certain when I put in a tendon graft at what tension

it should be done. If it is put in at too much tension, the finger cannot be straightened. If it is put in at too little tension, the finger cannot be completely flexed. Of the two faults I think that inability to flex is more serious than inability to extend the fingers completely.

I have great admiration for Dr. Burbank's and Dr. Marble's nerve grafts. We have not been so successful with our few nerve grafts. I would like to emphasize a point that Dr. Marble made on the application of pedicle flaps to fingertips. They must be thin. I have had the experience—and I expect that Dr. Marble has, too—of having to remove a pedicle flap after it has been applied because it is floppy and does not hug the tip of the finger tightly. The patient becomes embrasssed because it looks peculiar; but when it is put on thin, using skin for example from underneath the breast, we have succeeded in applying a graft with which the patient is satisfied.

I want to say a word about Dr. Carroll's presentation of cysts of the distal phalanx. We have seen, among others, one such case which was mistakenly diagnosed as a felon and was treated in various ways including x-ray. The result was almost complete destruction of the distal phalanx plus severe x-ray dermatitis.

James Barrett Brown (St. Louis, Mo.): A suggestion might be made to try to clear up a mistaken idea that is frequently made and that Dr. Crikelair has made, i.e., that a split graft does not have any derma on it. A split graft (which is the term we originally applied to it from having likened the process to splitting scraps of leather in a harness shop), is simply part of the skin split at any level you wish but it always contains some derma. One cannot actually split the skin without having some derma on it unless for some reason a graft of just epithelium might be wanted and it would be so thin it could hardly be handled. If we do ever mean to use such thin grafts it would be better to call them Thiersch grafts, yet even these Thiersch grafts have some derma on them.

The process is a method of splitting the skin, and the splitting takes place through the derma leaving part of it behind to allow for healing of the donor site. The derma of the back skin is several times thicker than it is on the palm, while on the palm the epidermis is a keratinized layer several times thicker than that on the back. This is important to remember in dealing with large burns because more than one crop of skin can be gotten from one area. Instead of discussing these papers, which have been most interesting, I would like to leave the idea that split skin grafts do have derma on them up to three-fourths of the whole thickness of the skin. Derma is the essential part of a split graft or of any graft. It is what takes the trauma and shock of getting around. Full-thickness grafts are important to use for certain reasons but not because a split graft does not contain derma.

L. H. McKim (Montreal, Canada): I would like to speak particularly with regard to the repair of trauma to the distal phalanx. Dr. Mason has spoken a very fine truth when he mentioned that the simplest way of doing anything is often the best way. Now there is a very simple method in guillotine amputations of the terminal phalanx that will work, especially where not too much has been removed. All that is needed is a small piece of x-ray film sterilized by sticking it into alcohol. After the finger has been cleaned you wrap that carefully around the finger, sloping it down toward the end and fastening it with adhesive. The wound is dressed through the end of that. Those who have tried it find that about 3 mm. are added to the length of the finger. Many of these amputations will very easily epithelize without your doing any flaps or any other repair. I mention this merely as a simple method which can be carried out in a very short time and which is frequently used by the general practitioner with great success.

Carleton Mathewson, Jr. (San Francisco, Calif.): I should like to discuss Dr. Crikelair's paper on severe trauma to distal digital extremities.

In our experience crushing injuries to the fingers are very common in childhood, usually as a result of having a finger crushed in an automobile door. Nothing was said about anesthesia or the methods of cleaning these wounds. We have been so unfortunate in the past year to have lost, as the result of anesthesia, two tiny children who had crushed their fingers in a car door. In both these cases a period of over six hours was waited and the stomach emptied by lavage before anesthesia was introduced. Regardless of these precautions, both of these children died as a result of aspiration. Both had expert anesthesiologists.

We believe that it is extremely important to avoid general anesthesia as an emergency procedure in tiny children. We have found that often a small amount of nerve block anesthesia is sufficient to handle these injuries. In older children (and adults) we can anesthetize their extremity with local block anesthesia and then take them to the scrubroom with us and make a game of scrubbing the extremity thoroughly with soap and water.

I would like to emphasize the importance of cleansing the hand thoroughly. I have watched the initial cleansing in many clinics. It is usually undertaken by a junior member of the staff or a nurse and it is usually done, to my way of thinking, improperly. Too frequently one depends on some sort of antiseptic to cleanse the hand rather than thorough scrubbing with soap and water. Often the major portion of the procedure, when you receive these injuries early, is proper local anesthesia and thorough cleansing with soap and water.

In regard to repair we have found that in the great majority of crushing wounds they appear to be much worse than they really are. It is unfortunate that many of these digits are amputated because of their appearance. There are a large amount of hemorrhage, subcutaneous hemorrhage and a good deal of swelling. The finger usually explodes, so to speak, but there is usually enough epithelium remaining between these areas of explosion for healing. Often we have not even had to resort to suture. Actually after very thorough cleansing we often can use sterile adhesive tape for approximation, which is not painful.

One other point which I would like to emphasize is that it is extremely useful when a fingernail remains in place; even though you realize that you are going to lose it eventually, it should be left in place after thorough cleansing as a dressing. It covers the nail bed and is the best possible dressing you can have.

If these patients have avulsion of skin or if they bring in the tip of their finger, let me once again emphasize the importance of removing all of the fat before you replace the skin. If you do not, you will lose the graft.

Next to cleansing, the most important factor in treatment is immobilization. Reparative surgery usually fails when the débridement and immobilization are inadequate.

GEORGE F. CRIKELAIR (closing): We agree with Dr. Mathewson and have been following the procedure that he outlined for children. We no longer subject any of them to general anesthesia. We find that we can do just as well treating them quite conservatively. His points of immobilization and cleansing are paramount in any hand injury.

I would like to thank Dr. Brown and Dr. Mason for clearing up some technical points. The pedicle flap to the fingertip is pointed out by Dr. Marble as generally unsatisfactory because it is usually too fat. Also, sensation is slow in returning and there is usually some pigmentation. However, there are a few cases in which it does seem to be indic ted and it might as well be done as a primary procedure.

ROBERT E. CARROLL (closing): I would like to thank the discussers for their helpful suggestions. As far as Dr. Marble's suggestion of the glomus tumor, I must say that the size in itself of the glomus tumor as I have seen it is about the size of a grain of rice. I have not seen a fracture through a glomus tumor, even in those which have had pressure on them. Often this neurovascular tumor is incorporated with the type of tumor which is extrinsically pressed into bone, and not incorporated within the bone itself. I think this is true.

Dr. Mason's comment upon the treatment is not unusual. We now have about a dozen such cases of tumor in the finger which have had the most bizarre types of treatment. It is purely because the most fundamental thing we have is the diagnosis, and this is incorrect. Therefore treatment was given before the diagnosis. We can hope to reverse this.

# CONSERVATIVE MANAGEMENT OF LOW BACK PAIN

B. E. McConville, M.D.

*Seattle, Washington*

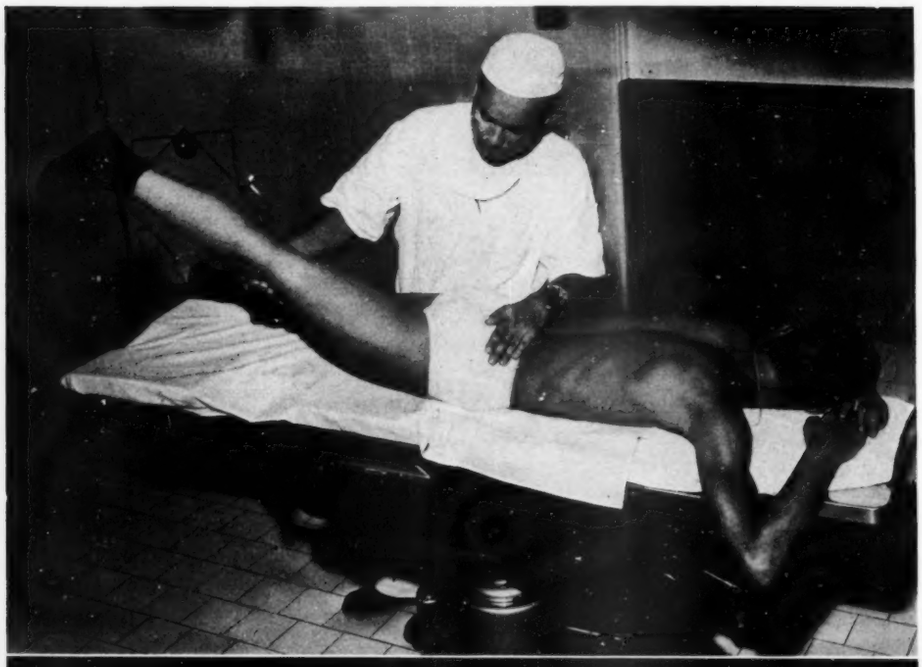
LOW back pain continues to be a major problem in industry and presents a continued challenge to the attending physician. There are many causes of low back pain, and a careful history and physical examination are necessary to establish a diagnosis. For the purpose of this discussion only the low back pain resulting from injury will be considered. With the impetus by Dandy,[1] Barr and Mixter[2] and others, the popularization of the protruded lumbar disc has overshadowed other causes of low back pain seemingly almost to their exclusion and has resulted in a marked increase of spinal surgery. It is the purpose of this paper to focus attention on other injuries causing low back pain and to emphasize the conservative treatment for low back injuries. Despite the claims of individual operators, surgery of the spine has been disappointing in the over-all aspect and particularly so in industry because of the high degree of permanent disability resulting. The Washington Rehabilitation Center, treating only industrial cases, reports only one patient of three returning to his former occupation. Not only is the average permanent partial disability rating of surgically treated low back injuries high but also it is one of the major causes of pension in the state of Washington. Certainly there are definite indications for spinal surgery, but only after a trial of conservative treatment. Surgical emergency of the low back is indeed rare. In the majority of cases conservative treatment of low back injuries is successful and there is less permanent disability.

In the category of conservative treatment manipulation of the back is a most valuable aid. Although manipulation of the back has frequently been described in the literature[3–7] and its merits enthusiastically proclaimed, it has failed to achieve popularity probably because of a possible stigma associated with unorthodox practitioners. It is a safe procedure and when properly used does not entail any particular hazard. Back manipulation under anesthesia can be employed successfully in many types of low back pain and should be given a trial even when a definite diagnosis of protruded intervertebral disc has been made. The number of patients who will not need surgery is rather remarkable. There are several methods of manipulation. A complete description of manipulation by the rotation method can be found in excellent articles by the Doctors Travell[3] and Schaubel.[7] We have used two variations in our series, one with the patient in a prone position (Fig. 1) with hyperextension and abduction of the affected leg with pressure over the sacroiliac area, followed by general hyperextension of the spine. In the second method the patient is in the supine position with the shoulders lying flat on the table. The pelvis is rotated with the affected side upward and with the knee and hip flexed. The right hand grasps the affected leg above the knee and uses this as a lever for manipulation with pressure being brought downward on the leg. At the same time the shoulder is braced with the left hand. (Fig. 2.) Usually an audible snap can be heard during the manipulation. Back manipulations at the hospital were carried out under sodium pentothal. Less severe cases of low back pain can be treated by back manipulation as an office procedure without anesthesia.

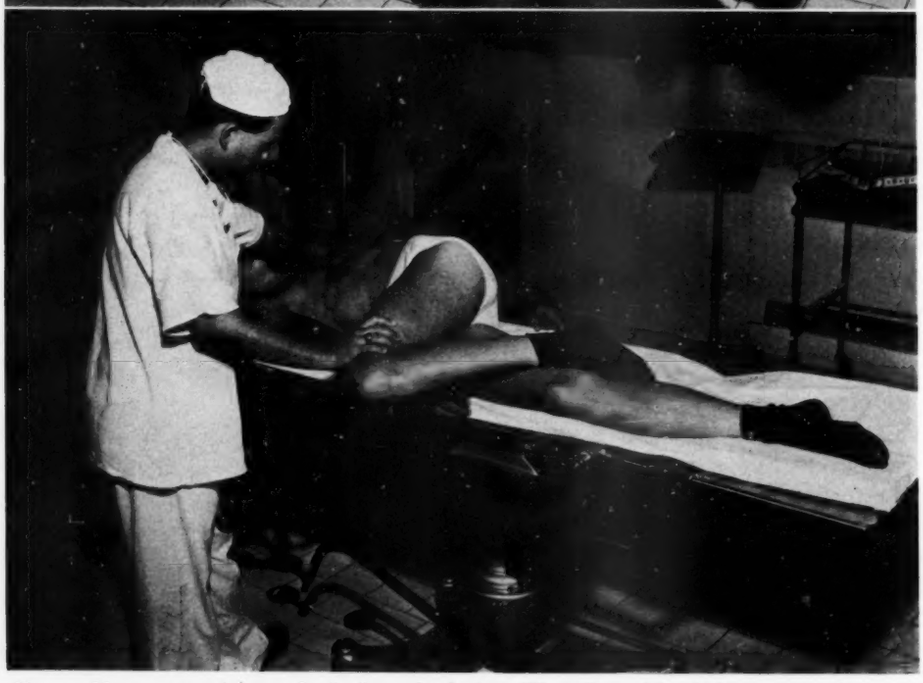
Epidural injection using normal saline is another valuable aid, particularly in those cases of low back pain associated with sciatica. An excellent description of the technic can be found in the article by Evans.[8] An epidural injection should always follow back manipulation and not precede it. The complication of retinal hemorrhage, although very infrequent, should be borne in mind.

Other conservative measures such as bedrest, leg traction, physiotherapy, the use of muscle relaxants such as tubocurarine and the use of intravenous salicylates (which we believe have a definite clinical advantage over oral salicylates) have a definite place in the over-all management of all low back pain.

A differential diagnosis should always be thoroughly considered before a definite diagnosis is made. Time will not permit a complete differentiation; however, the majority of cases will fall in the category of lumbosacral facet



1



2

FIG. 1. Hyperextension method of manipulation: The affected leg is hyperextended and slightly abducted with pressure over the sacroiliac area.

FIG. 2. Rotation method of manipulation: The shoulders lie flat on the table with the pelvis rotated and with the affected side upward. The upper knee and hip are flexed using the affected leg as a lever with pressure being brought downward on the leg and at the same time bracing the shoulder.

syndrome or sacroiliac displacement and that of the protruded intervertebral disc. The diagnostic points of a protruded intervertebral disc have been frequently described and in my opinion should always present a definite picture of sensory or motor nerve disturbance.[9] The lumbosacral facet syndrome has been advanced as a definite clinical entity based on the supposition that there is an infolding of the synovial membrane between the articulating facets. Clinically, I find no variation between this and sacroiliac displacement,[10–12] a term which for some reason has fallen in some disrepute possibly because it is commonly employed by non-medical practitioners. Consequently, I shall consider lumbosacral facet syndrome and sacroiliac displacement as the same condition. The pertinent points are as follows: In the standing position there is a list of the spine away from the affected side. There is marked lumbar spasm with the affected side less prominent. The point of tenderness on palpation is over the posterior iliac spine. There is definite splinting of the affected side on all motions, with pain particularly noted on flexion and on lateral motion to the affected side. In the supine position measurements of the legs taken from the anterior superior spine to the medial malleoli are equal; however, and this is an important point, there is an apparent lengthening of the affected side by comparison of the level of the internal malleoli. This, in my opinion, is a main differential point. Straight leg–raising on the affected side is painful while that of the opposite causes little distress but may be referred to the involved side. Patrick's and Gaenslen's tests on the affected side are positive and negative on the opposite. Torsion or rotation of the pelvis on the spine is definitely positive with the patient pointing to the sacroiliac area. There is no sensory disturbance. The reflexes are present and equal bilaterally. There is usually no muscular atrophy unless the condition has persisted for a considerable period of time which then usually is on the basis of disuse atrophy. X-rays should always be taken of the lumbosacral area to rule out definite bony disease or abnormalities. The history of onset is usually a twist, bend or a lift from an off-center position.

During the past five years, 261 patients have been treated with manipulation under anesthesia. Of these, 153 had sciatic nerve pain. Only forty-five required a pantopaque study and only twenty-nine required laminectomy. The average hospitalization of this series was nineteen days. The shortest period of hospitalization was two days and the longest period 126 days. The average duration of symptoms prior to hospitalization fell into three separate time intervals: 60 per cent of the series averaged five months, 22 per cent averaged two and a half years and 18 per cent averaged two weeks. Six cases were not included in these averages; one patient had a duration of five days, another of seven years, three of ten years, and one of fifteen years. The average postoperative office treatment was seventy-one days. The average time loss on the industrial cases which could be obtained from the records was eighty-one days. Of this series, 73 per cent were men. The age groups were as follows:

| Age (yr.) | Per cent |
|---|---|
| 20 to 29 | 15 |
| 30 to 39 | 37 |
| 40 to 49 | 22 |
| 50 to 59 | 22 |
| 60 to 69 | 4 |

Not included in the age groups was one patient aged eighteen and another aged seventy-six. Of the entire series forty-six required a second manipulation and twenty-one required a third. Epidural injections were given to all patients with sciatic radiation.

## CONCLUSIONS

1. A thorough trial of conservative treatment should be followed before back surgery is attempted.

2. Manipulation of the back under anesthesia will materially reduce the number of patients requiring spinal surgery.

3. Epidural injection is a valuable aid particularly in those cases in which there is sciatic nerve pain.

4. Two hundred sixty-one patients were treated by manipulation under anesthesia and only twenty-nine required a subsequent laminectomy.

## REFERENCES

1. Dandy, W. E. Concealed ruptured intervertebral discs. A plea for the elimination of contrast mediums in diagnosis. *J. A. M. A.*, 117: 821–823, 1941; Treatment of recurring attacks of low backache without sciatica. *J. A. M. A.*, 125: 1175, 1944.

2. Mixter, W. J. and Barr, J. S. Rupture of inter-

vertebral disc with involvement of spinal canal. *New England J. Med.*, 211: 210, 1934; BARR, J. S. and MIXTER, W. J. Posterior protrusion of the lumbar intervertebral discs. *J. Bone & Joint Surg.*, 23: 444–456, 1941.

3. TRAVELL, W. and TRAVELL, J. Technique for reduction and ambulatory treatment of sacroiliac displacement. *Arch. Phys. Therap.*, 23: 222, 1942; TRAVELL, J., and TRAVELL, W. Therapy of low back pain by manipulation and of referred pain in the lower extremity by procaine infiltration. *Arch. Phys. Med.*, 27: 537–547, 1946.
4. BANKART, A. S. B. The use and abuse of manipulative surgery. *Brit. M. J.*, 2: 416–418, 1936; Low back pain and sciatica. *Practitioner*, 157: 367, 1946.
5. FINDER, J. G. Manipulative surgery in orthopedics. *S. Clin. North America*, pp. 243–259, 1949.
6. JOSTES, T. A. Backache. Manipulative treatment without anesthesia. *J. Bone & Joint Surg.*, 20: 990–1010, 1938; PITKIN, H. C. Sacroarthrogenetic telalgia; plan for treatment. *J. Bone & Joint Surg.*, 19: 169–18, 1937.
7. SCHAUBEL, H. J. The manipulative treatment of low back pain. *J. Michigan State M. Soc.*, 49: 913, 1950.
8. EVANS, W. Intrasacral epidural injection in the treatment of sciatica. *Lancet*, 2: 1225, 1930.
9. KEEGAN, J. J. Dermatome hypalgesia associated with herniation of intervertebral disc. *Arch. Neurol. & Psychiat.*, 50: 67–83, 1943.
10. ALBEE, F. H. Study of the anatomy and clinical importance of the sacroiliac joint. *J. A. M. A.*, 153: 1273, 1909.
11. CYRIAX, E. Minor displacements of the sacroiliac joints. *Brit. J. Phys. Med.*, 8: 191, 1934.
12. GRAY, H. Sacro-iliac joint pain; finer anatomy; mobility and axes of rotation; etiology, diagnosis and treatment by manipulation. *Internat. Clin.*, 2: 54–96, 1938.

# MANAGEMENT OF LOW BACK AND SCIATIC PAIN*

G. Edmund Haggart, M.D. and William R. Grannis, M.D.
*Boston, Massachusetts*

PAIN in the low back and radiating into one or both lower extremities continues to present a difficult diagnostic problem, as is attested by the voluminous literature on this subject. Steindler[17] aptly sums up the situation when he states that "backache shares with the symptoms of headache the distinction of having almost unlimited possibilities of interpretation." Barr[2] stresses the fact that the low back is an extraordinarily complicated mechanism and that its anatomy, physiology and pathology are still imperfectly understood, as is well documented by an imposing list of potential causes of low back and sciatic pain.

The object of this paper is to summarize our plan of management for patients with low back and sciatic pain; to emphasize there is no one common etiologic factor; and in particular, to stress the value of adequate conservative therapy as well as to describe briefly procedures that are of proven value in deciding on the diagnosis and subsequent treatment.

In a two-year period (1946 and 1947) 6,402 patients were examined because of back and/or sciatic pain. Of this number, 322 consecutive patients with severe symptoms, or 5 per cent, were hospitalized and are the basis for this report. Follow-up data were obtained by personal examination of 171 patients and by answers to a detailed questionnaire by twenty-nine patients who were unable to report at the clinic in response to our request.

This method of selection provided for the study of patients with significant disability, as indicated by their acceptance of hospitalization, and for variety in their age, sex, economic level and occupation. This group also minimized the selection of patients in terms of the quality of the results obtained by treatment. In the patients reviewed herein symptoms are believed due to a mechanical or physiologic derangement of the motor-skeletal system subsequent to static strain; following degenerative changes, including the disc syndrome, or due to an unstable lumbosacral spine, the result of structural anomalies or following injury. Not infrequently a combination of these factors was present. Neoplasm, inflammatory diseases including the specific arthritides, as well as intrapelvic and intra-abdominal pathologic conditions, were always considered in the differential diagnosis.

## DIAGNOSIS

Low back and sciatic pain consistently presents a major diagnostic problem which demands an exhaustive history and analysis of all factors, traumatic and otherwise, in the patient's environment, as well as data in the family history that may shed light on his physical and mental status. In addition to studies by the orthopedic surgeon the physical examination includes a review by the medical service as well as by the neurologic consultant. It is essential to consider the actual degree of disability imposed by the patient's symptoms in the light of his particular environment and objectives when deciding upon the form of treatment. It is particularly important that the examiner appreciates the significance of faulty body mechanics and the aggravation of symptoms imposed by overweight and inadequate musculature as well as by degenerative or traumatic skeletal changes.

The initial roentgenograms comprise a lumbosacral series including a 45 degree angle anteroposterior projection to delineate further the sacroiliac and lumbosacral articulations, as well as right and left oblique films for additional visualization of the articular facets in the lower lumbar spine. Forward and lateral flexion and hyperextension roentgenograms are secured in cases of suspected spondylolysis or when there is a question of pseudarthrosis in a previously performed spine fusion.

We do not believe that the presence of a narrowed disc space in the roentgenogram is necessarily of significance in making a diagnosis of pathologic condition of the disc.

At this stage and with the foregoing data an initial evaluation of the case is made, not only as to possible organic basis for symptoms but

* From the Department of Orthopedic Surgery, The Lahey Clinic, Boston, Mass.

also, in particular, a survey of the patient's emotional stability. Compensation cases require additional detailed screening. The results of the medical and neurologic examinations are of great assistance in deciding whether there are pathologic changes present not usually classified under an orthopedic heading, and also as an aid in evaluating the individual. We wish to stress this aspect of the study because such a comprehensive survey of the patient is of marked significance in deciding on the initial treatment.

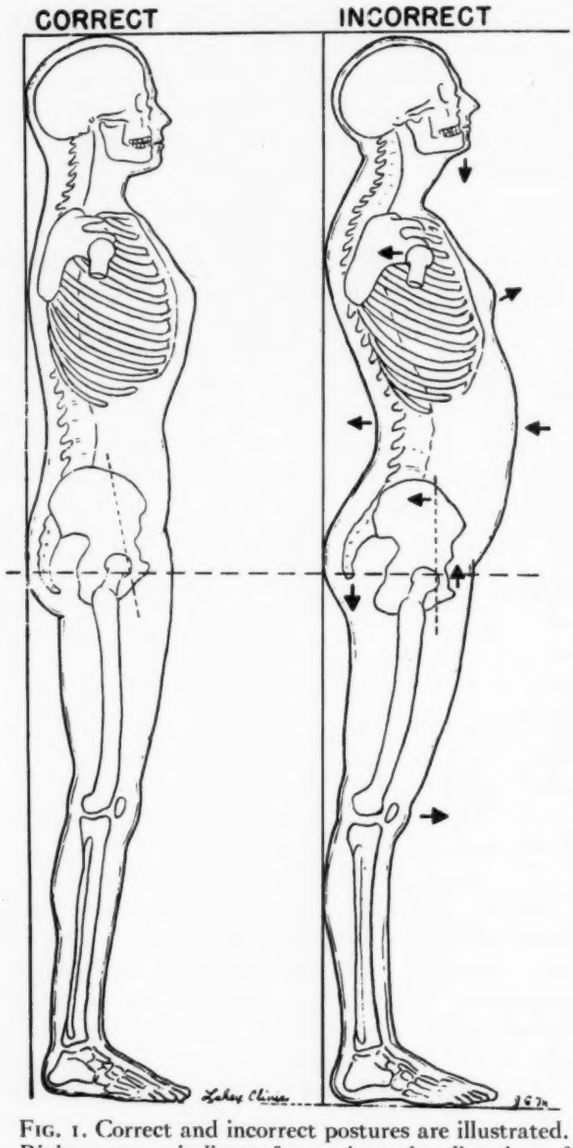



FIG. 1. Correct and incorrect postures are illustrated. Right, arrows indicate for patient the direction of muscle action to correct postural faults (see text).

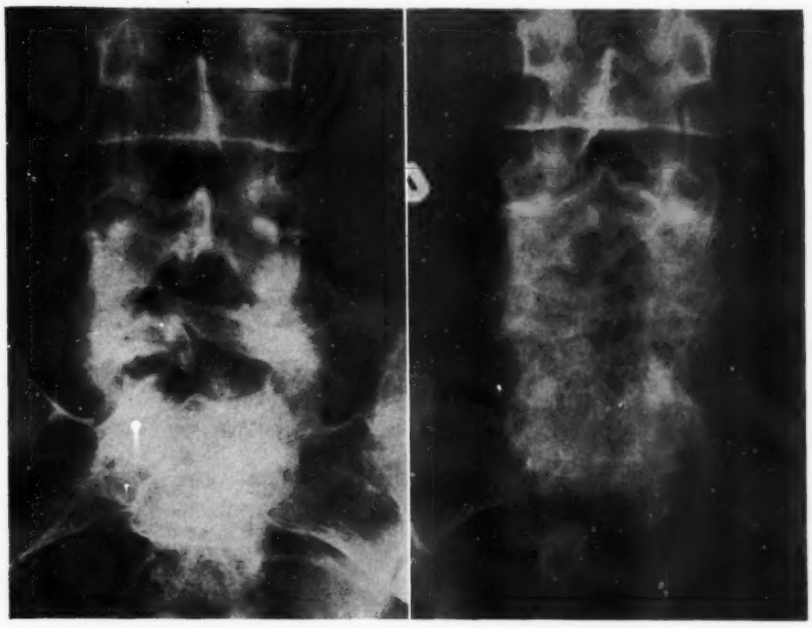
Pain that radiates down the sciatic distribution indicates direct nerve or root involvement. Such symptoms are most frequently caused by a pathologic condition of the disc which is usually due to degenerative changes within the disc but may be associated with or aggravated by trauma. However, leg pain simulating true sciatic pain can occur from other causes, as observed for example in cases of sacroiliac arthritis or in certain lesions in the pelvis. Evidence of a radicular lesion can often be demonstrated by careful examination, with particular reference to the sensory and motor distribution of the nerve roots.

So-called sclerotogenous pain[10] is not associated with well localized motor and sensory deficits caused by a radicular lesion and the deep tendon reflexes are not markedly altered. The differential is not always clear-cut, however; hence we have come to rely on pantopaque myelography which we routinely employ when the differential diagnosis includes a possible pathologic condition in the spinal canal including disc protrusion. As indicated in a recent communication,[8] this particular examination is usually performed without complications and in the majority of cases gives definitive information, namely, accurate localization of disc protrusions as well as demonstrating the presence or absence of other intraspinal conditions. The myelogram often makes possible an exploration limited to the disc space indicated so that disc surgery will upset the mechanics of the lower spine to a minimal degree.

In our experience hypertrophy of the ligamentum flavum has been of relatively minor significance. The condition has been observed, but in the majority of cases was not considered to be of special clinical importance.

## CONSERVATIVE TREATMENT

An aspect of treatment applicable to all patients and which represents the principal point we wish to stress concerns the use of exercises. Frequently a patient protests against such a program, stating that he has tried exercises and thereby experienced marked aggravation of symptoms. However, the patient's attention is directed to the position of his pelvis and to those muscles that control pelvic motion. A copy of Figure 1 is given to each patient as it well illustrates his objectives and indicates the particular muscle groups that should be exercised. The patient is taught a kinesthetic



2A 2B

FIG. 2. This patient, a thirty-two year old chemist, had recurrent low back pain of two years' duration with left sciatic radiation for one year and progressive disability. A, transitional unstable type lumbosacral junction is shown with reactive changes at that level as well as between the unstable facets of the fourth and fifth lumbar vertebrae. At operation, May 5, 1947, disc protrusion was removed from the left fourth lumbar space, followed by fusion from that level to and including the sacrum. B, on August 11, 1952, there is firm fusion; patient is asymptomatic and physical examination is negative.

sense as regards the position of his pelvis as he stands, sits and reclines. The active exercises employed are principally muscle-setting exercises of the glutei and lower abdominal musculature acting to tilt the pelvis under the lumbar spine and performed in the various postures.

With the concept that the patient's pain is related to irritation of the joints and periarticular structures in the lower lumbar spine and the muscle spasm resulting therefrom, this irritation tends to be relieved as the lumbosacral angle is diminished by maintaining the pelvis in improved relationship to the spine. Furthermore, it is reasonable to believe that as the space is increased between the posterior elements of the low spine by correcting an acute lumbosacral angle, local diminution of edema and circulatory disturbance in this area follows.

When there is muscle spasm in the low back which "sets" the lumbosacral angle, exercises which demand hyperextension, forward bending or trunk rotation may transmit increased shearing force across the lumbosacral articulations and so aggravate the existing irritation. Therefore, the exercises advised are not designed to increase range of motion in the low back. In the majority of patients when the tone of these muscles which hold the pelvis in good relationship to the spine is sufficiently improved by these muscle-setting exercises, the back pain diminishes and the individual becomes more active and regains an increasing range of motion in the course of his normal activities.

These exercises are progressively performed even by those individuals with acute back symptoms necessitating complete bed rest, as well as in the postoperative phase after spinal surgery. Furthermore, we have repeatedly noted that the symptoms of ambulatory patients who have long been subject to intermittent low back episodes as seen in the "facet syndrome" can be controlled and the patients are free from disability often for an indefinite period by performance of these exercises at stated intervals during each day.

The patient is impressed that the exercises are his own responsibility and that he must not rely on massage of the back or treatment by various types of radiation therapy. When rehabilitation has progressed sufficiently, swim-

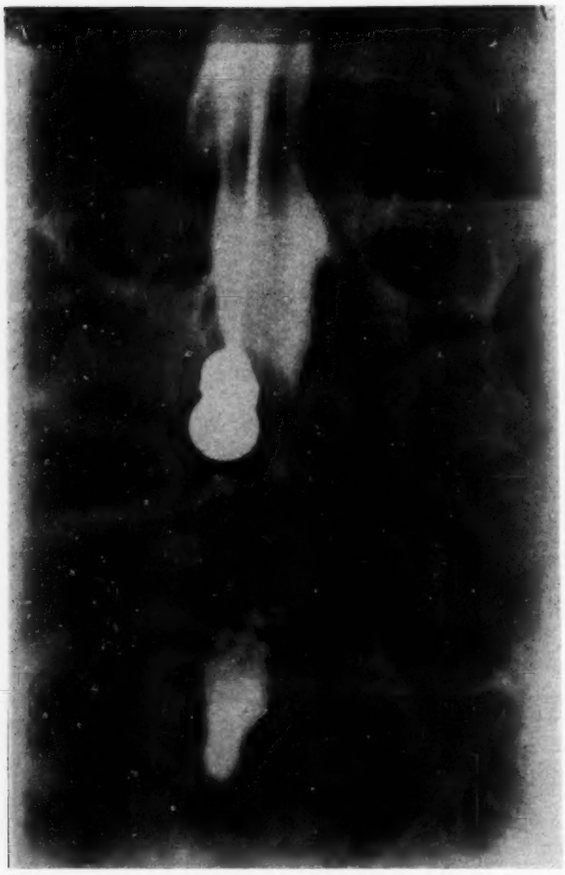


FIG. 3. A carpenter, age 50, for six months had progressive disability from severe left sciatic pain; incapacitated when first seen. Myelogram exhibits complete block at fourth lumbar space. At operation September 8, 1947, a very large, ruptured intervertebral disc was removed.

ming is strongly recommended as a most beneficial sport although diving and riding breakers are condemned.

While the concept and performance of these exercises seem simple, indeed, many patients have difficulty in acquiring the knack. Our physiotherapists have been of immeasurable aid in personally supervising each patient until he acquires the ability to contract the proper muscles in the proper manner.

In those individuals with relatively minimal symptoms and no real disability or in the group of patients with a history of recurrent low back and leg pain and who are seen when symptoms are mild or minimal, attention is directed to faulty body mechanics, with particular stress on the exercises previously described. When indicated, weight reduction, the use of bed boards and corollary medical attention as required are instituted.

In the group of patients whose symptoms and findings indicate definite disability at the first visit, hospitalization to initiate treatment is advised. In many of these patients the acute symptoms subside promptly with rest and sedation. Postural and muscle training is begun while they remain at bed rest. They are then graduated to an ambulatory status and continue a progressive exercise program.

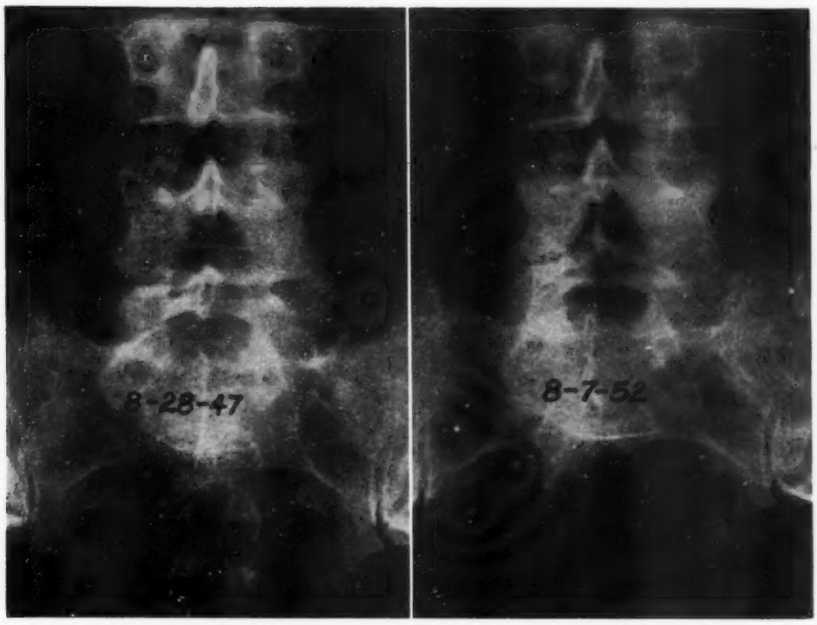
In patients who are incapacitated or in those individuals in the second and third groups whose symptoms fail to subside with initial bed rest, particularly if there is sciatic radiation of pain, a pantopaque myelogram is performed in the hospital. Immediately thereafter the patient continues on complete bed rest with exercises and, depending on the roentgenographic findings, is either advised of surgery or given a further period of conservative treatment.

In summary, therefore, the majority of patients with low back and sciatic pain are initially treated on a conservative basis. More radical therapy is indicated only for individuals who do not respond to such treatment or who are incapacitated when first seen. If the symptoms subside relatively rapidly and the patient gradually becomes ambulatory and carries on this program, it may be effective for many months or years; this is especially true if the symptoms described comprise an initial attack.

In patients who continue to exhibit involuntary muscle spasm, marked limitation of motion and sciatic pain following the myelogram all the findings are re-evaluated and a decision is made on the details of operative treatment.

## OPERATIVE TREATMENT

Whether or not fusion is performed with disc excision depends primarily upon the history and on the type of bone anatomy shown on the preoperative roentgenograms of the spine. A fusion procedure is indicated if these latter films reveal an unstable type of articular facets with reactive bone changes (Fig. 2) and, in turn, the individual has experienced back pain for a considerable interval which may antedate the onset of sciatic pain. On the other hand, fusion is not warranted in a patient incapacitated by sciatic pain with a positive myelogram (Fig. 3) but who exhibits widespread degenerative changes. (Fig. 4.) A fusion operation is not performed if the low back symptoms are relatively mild and the roentgenographic examina-




4A 4B

FIG. 4. A and B, pre- and postoperative roentgenograms. Degenerative arthritis of lumbosacral spine is evident on the admission films and it had increased when last seen, August 7, 1952. However, this patient returned to work five months after operation and has continued with his normal activities. Clinical examination negative. At the time of myelography it was not possible to withdraw the pantopaque distal to the obstruction. Note that in the postoperative film there is no evidence of opaque media.

tion is negative, and if at surgery the pathologic condition of the disc as demonstrated by myelography (Fig. 5) is revealed as a large disc protrusion or an actual herniation.

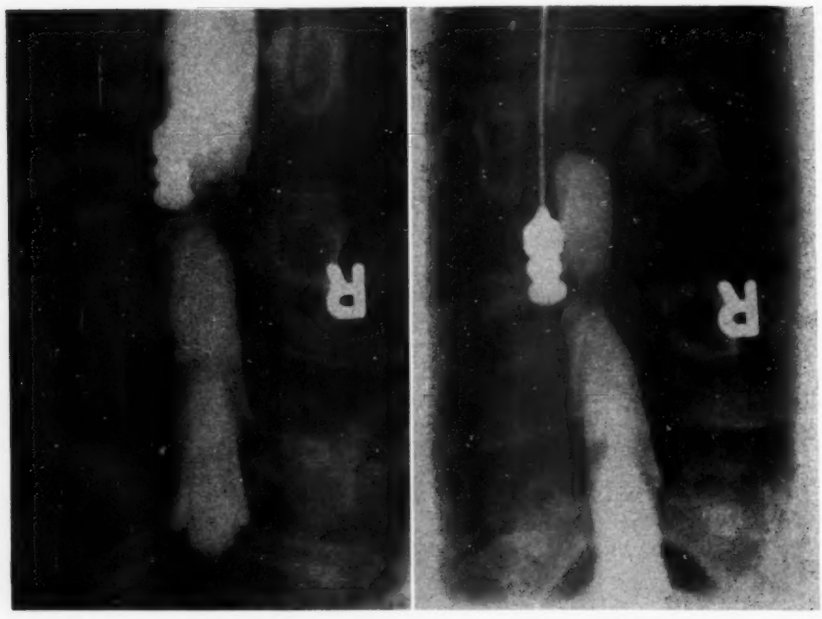
Evaluation of the patient's personality, as described heretofore, is also of assistance in the decision regarding a fusion procedure. In the unstable individual who exhibits definite clinical findings of a disc protrusion, and especially with intractable sciatic pain, surgery is limited to disc excision. Even if the data would ordinarily indicate a fusion, the latter operation is rarely performed upon this type of individual.

Traction applied to the lower extremities of patients with low back and sciatic pain is, we believe, relatively ineffectual. Either their symptoms will subside on complete bed rest, combined with the muscle-training program as noted, or pain and disability persist. If the latter is the case, the next step is the myelogram followed by a decision as to surgery.

Manipulation of the back under anesthesia has occasionally been employed when it was our opinion that the symptoms were on the basis of fibrositis or periarticular fibrosis. Such a diagnosis first demands the exclusion of disc disease by myelography, and neoplasm or destructive inflammatory disease. Carefully selected patients have been helped by manipulation, but we have rarely considered this procedure the treatment of choice. Occasionally a valuable adjunct is a straight leg–raising maneuver under anesthesia postoperatively in disc patients who have persistent sciatic pain in whom a recurrent disc protrusion has been ruled out by a myelogram.

*Complications Following Surgery.* The mortality rate in either simple disc excision or excision plus fusion is very low. In the 322 patients reviewed herein there were no deaths, and in the series reported throughout the country the mortality rate has been consistently less than 1 per cent.

Postoperative infections may be divided into two types, with reference to whether the infection is superficial or deep, the latter involving the entire wound. In the former it is readily handled. In the latter and more serious infection diagnosis can be established within the first six to eight days following the operation. For such serious, deep-seated infections we strongly favor exposure of the entire operative wound,



5A 5B

FIG. 5. A and B, a thirty-four year old trucker, had recurrent, mild, low back and intense sciatic pain of two years' duration, finally incapacitating. Pantopaque myelogram (B) illustrates a definite defect at the third lumbar space that was due to an old, extruded mass of degenerated disc tissue which was adherent to the adjacent nerve root. Additional degenerated disc material was also removed from the space at operation on October 3, 1947. At follow-up study August 5, 1952, examination was negative; the patient was completely active and asymptomatic except for occasional slight stiffness of back.

packing it open, and institution of intensive antibiotic therapy. Bacterial sensitivity tests are important in deciding on the antibiotic. If a fusion procedure has been done, all of the bone grafts are removed at the time of the initial exploration. With the infection under control, a secondary wound closure is performed. In 200 cases superficial infection was noted in three patients. Deep infection of the wound occurred in one patient. All the wounds healed.

Thrombophlebitis was observed in one patient of the group. This low incidence may be attributed to the early resumption of the muscle-training program which the patient had been taught before surgery was performed.

*Postoperative Care.* In all patients the postoperative care is extremely important. The most significant part is the program of intensive muscle training, as described, and this is reinstituted within the first week following operation. The value of the assistance given by a competent physiotherapist cannot be overestimated.

Braces and special corset supports are not employed except in the postoperative period following spine fusion where such support permits earlier ambulation.

## RESULTS

The results in the 200 patients who had follow-up studies after five to six years were classified in the following manner: excellent when the patient expressed enthusiasm regarding his treatment and demonstrated no evidence of disability; good when the patient presented mild complaints of occasional symptoms but suffered no significant disability affecting his social life or occupation; fair when the patient admitted improvement but had sufficient residual symptoms to restrict his activities or grossly change his manner of livelihood; poor when the patient denied significant improvement or remained disabled for work or ordinary activity.

In the entire group the results were excellent or good in 182 patients or 91 per cent, fair in six or 3 per cent, and poor in twelve or 6 per cent. Disc operations were performed in ninety-seven cases or 48.5 per cent (disc exci-

sion alone in ninety or 45 per cent; negative exploration for disc disease in three or 1.5 per cent, two of whom achieved an excellent and one a poor result; nerve root ganglion in one or 0.5 per cent; and recurrent disc protrusions in three or 1.5 per cent). Combined disc excision and fusion was carried out in fifty-four or 27 per cent, and spine fusion was performed in thirty-eight or 19 per cent. Twelve of this number had exploratory operations for possible disc disease, with negative findings. In eleven patients or 5.5 per cent no operation was performed.

*Patients Having Disc Operations Alone.* The results in this group of ninety-seven patients were excellent in 71.1 per cent, good in 19.5 per cent, fair in 5.2 per cent and poor in 4.1 per cent. Two cases of superficial wound infection and one instance of thrombophlebitis constituted the complications in this group.

The incidence of disc protrusion at the various levels was in accord with the figures generally accepted, being most commonly encountered at the fourth lumbar space. Three patients or 3 per cent had recurrent disc protrusion necessitating reoperation.

*Patients Having Combined Disc Excision and Fusion Procedures.* The results in this group of fifty-four patients were excellent in 70.4 per cent, good in 27.7 per cent and poor in one or 1.9 per cent; in none were the results estimated to be fair.

Sixty-three per cent of this group of patients had fusions from the fourth lumbar down to and including the sacrum; lumbosacral fusion was performed in 35 per cent and in 2 per cent from the third lumbar vertebra to and including the sacrum.

*Patients Having Fusion Operations Alone.* The results in this group of thirty-eight patients were excellent in 76.3 per cent, good in 13.2 per cent and poor in 10.5 per cent; in none were the results estimated to be fair. Deep infection occurred in one of these patients but this subsided and healing resulted following the plan of treatment described.

## CONCLUSIONS

It is to be emphasized that while the data presented primarily refer to operated cases, these patients comprise only 5 per cent of the total number of 6,402 patients seen. All of these patients were hospitalized because of low back and/or sciatic pain. They do include the most difficult and resistant cases but the remaining patients who were not operated upon made such satisfactory progress that surgery was not performed. In this period of a minimum five-year follow-up they have remained sufficiently asymptomatic to the point that there would be no indication for their readmission to the hospital.

Consequently, it is our belief that the conservative plan of treatment as outlined herein is adequate to achieve an excellent to good result in the great majority of patients with low back and/or sciatic pain. Furthermore, in the small group requiring surgery the auxiliary conservative measures, particularly the exercises, contribute markedly to the results obtained.

*Acknowledgment:* The authors wish to express their appreciation to Miss Gracia Halladay, senior nurse in the Department of Orthopedic Surgery, for her assistance in correlating the data for this study.

## REFERENCES

1. Aitken, A. P. Rupture of the intervertebral disc in industry. Further observations on the end results. *Am. J. Surg.*, 84: 261–267, 1952.
2. Barr, J. S. Low-back and sciatic pain; results of treatment. *J. Bone & Joint Surg.*, 33-A: 633–649, 1951.
3. Camp, J. D. Contrast myelography past and present; Carman lecture. *Radiology*, 54: 477–505, 1950.
4. Ferguson, A. B. The clinical and roentgenographic interpretation of lumbosacral anomalies. *Radiology*, 22: 548–558, 1934.
5. Friberg, S. Anatomical studies on lumbar disc degeneration. *Acta orthop. Scandinav.*, 17: 224–230, 1948.
6. Ghormley, R. K., Love, J. G. and Young, H. H. "Combined operation" in low back and sciatic pain. *J. A. M. A.*, 120: 1171–1176, 1942.
7. Haggart, G. E. Early operation (spine fusion) in unstable lumbosacral joints. *J. A. M. A.*, 115: 2129–2132, 1940.
8. Haggart, G. E. and Grannis, W. R. Pantopaque myelography in low back and sciatic pain. *S. Clin. North America*, 32: 695–703, 1952.
9. Hampton, A. O. and Robinson, J. M. Roentgenographic demonstration of rupture of intervertebral disc into the spinal canal after the injection of lipiodol, with special reference to unilateral lumbar lesion accompanied by low back pain with "sciatic" radiation. *Am. J. Roentgenol.*, 36: 782–803, 1936.
10. Inman, V. T. and Saunders, J. B. deC. M. Referred pain from skeletal structures. *J. Nerv. & Ment. Dis.*, 99: 660–667, 1944.
11. Kubik, C. S. and Hampton, A. O. Removal of

iodized oil by lumbar puncture. *New England J. Med.*, 224: 455–457, 1941.
12. Mixter, W. J. and Barr, J. S. Rupture of the intervertebral disc with involvement of the spinal canal. *New England J. Med.*, 211: 210–215, 1934.
13. O'Dell, R. T. and Key, J. A. Results of the operative treatment of ruptured intervertebral disks. Instructional Course Lectures No. 11. American Academy of Orthopedic Surgeons. Ann Arbor, 1949. J. W. Edwards Co.
14. Poppen, J. L. The herniated intervertebral disk; au analysis of 400 verified cases. *New England J. Med.*, 232: 211–215, 1945.
15. Roche, M. B. and Rowe, G. G. The incidence of separate neural arch and coincident bone variations; a summary. *J. Bone & Joint Surg.*, 34-A: 491–494, 1952.
16. Spurling, R. G. and Grantham, E. G. Ruptured intervertebral discs in lower lumbar regions. *Am. J. Surg.*, 75: 140–158, 1948.
17. Steindler, A. Diseases and Deformities of the Spine and Thorax, p. 293. St. Louis, 1929. C. V. Mosby Co.
18. Toumey, J. W., Poppen, J. L. and Hurley, M. T. Cauda equina tumors as a cause of the low-back syndrome. *J. Bone & Joint Surg.*, 32-A: 249–256, 1950.
19. Williams, P. C. The diagnoses and conservative management of lesions of the lumbosarcal spine. Lectures on Regional Orthopedic Surgery and Fundamental Orthopedic Problems, No. 11, pp. 103–116. American Academy of Orthopedic Surgeons. Ann Arbor, 1948. J. W. Edwards Co.
20. Willis, T. A. Low back pain; the anatomical structure of the lumbar region, including variations. *J. Bone & Joint Surg.*, 19: 745–748, 1937.

## DISCUSSION OF PAPERS BY DRS. McCONVILLE, AND HAGGART AND GRANNIS

Ralph K. Ghormley (Rochester, Minn.): I am in agreement with most of what has been said. I certainly believe an accurate diagnosis is the first requisite in any problem involving the lower part of the back. This is not always easy to make. There are various aids to diagnosis and one may have to resort to many of these in order to make an accurate diagnosis. Once it has been made, a conservative program of treatment is desirable.

As Dr. Haggart has pointed out, there are residual groups of cases in which conservative measures will not suffice. In these cases operations should be carried out with the utmost care and precision. I think that there has been too much operating in these cases in which patients complain of low back pain because we certainly are seeing a great many patients who have undergone one, even two and sometimes three operations without relief, and they present some of the most difficult problems we have encountered. It is almost impossible to decide just what to do in many of these instances. In some of these cases drug addiction is a complication, and in many the back is genuinely painful.

I think that in most cases in which severely disabled patients have undergone operations the greatest trouble is the formation of scar tissue around the operative site, and that scar tissue may extend to the nerve root. We have found it difficult to rehabilitate some of these patients and have been succeeding in some cases in which the patients have followed our program of carefully outlined exercises, which are painful, and have had the fortitude to cooperate. Once the scar tissue is mobilized, the pain becomes more bearable.

Cases of rheumatoid spondylitis comprise another group in which it is difficult to make an early diagnosis. In a group of cases studied at the Mayo Clinic some years ago it was found that in at least 10 per cent of the cases of rheumatoid spondylitis the first symptom was sciatic pain. In a number of these cases the patients came to the clinic after operation for protruded disc had failed to disclose this lesion.

There are many other phases of the problem. The diagnosis, as I said at the beginning, is most important. The outline of the program of treatment should be based, if possible, on a conservative program, and operative treatment should not be employed unless clearly indicated.

Alexander P. Aitken (Brookline, Mass.): It is indeed a pleasure to hear two consecutive papers pleading for the conservative treatment of low back pain, with or without sciatic radiation. You will recall that in 1947 I presented a paper before this group on the end results of disc surgery in industrial cases. Those results were very, very poor. That paper had some effect, I think, in dampening the enthusiasm for disc surgery for a short period of time. Since then, however, the same degree of enthusiasm has developed. As a result, we have made a study of an additional 200 patients operated upon between the years 1947 and 1949. It is understandable that with our inability to choose cases and to develop proper technics in such cases, when we first deal with them, our end results would not be good; however, with experience one would expect that we would be able to choose our cases better, develop a better technic and achieve satisfactory end results. In this last 200 cases, however, it has been disappointing to find that our end results have not improved, not one iota. They are just as bad now as they were in 1941 through 1947.

There are many reasons for this. Dr. Haggart has brought out the point of emotional instability, which is a very definite factor in the industrial case and most contributory to a poor end result, particularly in women. In our total series of 430 cases there were thirty-six women, and of these only four ever returned to any type of work.

The question of accurate diagnosis has been brought up. How do you make a diagnosis of a ruptured disc? I have actually seen, you may believe me, literally thousands of patients with back and leg pain, and I do not know yet how to make a diagnosis of a ruptured intervertebral disc. It is generally assumed if there are neurologic findings and if the patient has nervous irritation, you have a ruptured disc. In the last group of 200 there were thirty-five cases in which the surgeon admitted that he found no such condition. Of the thirty-five cases there were twenty-six that showed neurologic change, either in the form of sensory disturbance, atrophy or reflex changes. Of this group of twenty-six there were seven in which the triad of atrophy, reflex changes and sensory disturbances were all present. There were, in fact, two cases in which there was a toe drop, and yet at operation and exploration from the region of L1 through S1 nothing was found to account for the neurologic changes. You may say these were done by an inexperienced surgeon. They were not. They were done by individuals of reputation; they were done by capable men and yet they were unable to find the exact cause for these neurologic findings.

From my point of view the diagnosis of a ruptured disc is a most difficult diagnosis to make. Certainly it cannot be made on the basis of a single examination. I believe it can be made only after the patient has been given a period of conservative treatment: bed rest, boards, packs, deep x-ray, if you wish, manipulation and all the other modalities that we have at our command. However, we must stress conservative treatment. We must do everything we possibly can to see if the patient will not quiet down by himself. We must think of the large number of people in this country who at some time or other have back and leg pain. The total number of those people who should come to surgery, who actually have rupture of the intervertebral disc, is very small indeed, perhaps less than 5 per cent.

The point I wish to bring out and emphasize, as has been done by the previous speakers, is the importance of conservative treatment. Not only is it good treatment for low back pain but also, as far as I am concerned, it is one of the most important diagnostic tests that we have. Most people with back and leg pain will get better if given a chance. Only in those cases in which there is failure to respond to conservative treatment do we believe you are justified in performing a myelogram or surgery.

J. Huber Wagner (Pittsburgh, Pa.): Speaking after Dr. Aitken is somewhat of an anticlimax. He has given this subject great study and concern, and I must agree with everything he has said. I have always been for the conservative treatment of low back pain. I think many, many of these patients can be cured if you have enough patience with them and try to conserve them.

Further, I am very much in accord with the thoughts expressed by Dr. McConville so far as manipulative surgery is concerned in cases of chronic low back pain. The acute cases are different problems. We see many of these in our industry (the steel and coal industry) of which I have charge, and we have our own treatment for them with fair to good results. However, in the chronic cases we use manipulative surgery following the teachings of Bankart of England, who has been writing and studying this subject for twenty years. We have been following his lead on the method of treatment. I believe we get good results. We do a minimum amount of surgery.

I would like to say just the opposite of Dr. Haggart. I think that manipulation should take place before any spinal injections are made. Although pantopaque may be absorbable. I have seen many cases—several, I will put it—in which definite, prolonged reticulitis has been present. Some required surgery to remove the pantopaque before relief. I would therefore admonish you, if you have many of these cases and have not tried the manipulative type of surgery, that you do so. I think it well worthy of your efforts.

Nicholas James Giannestras (Cincinnati, Ohio): I want to thank Drs. Haggart and McConville for their excellent presentations on conservative treatment. However, there is one thing that I should like to ask Dr. McConville. How does he make a diagnosis of sacroiliac displacement? Is it done radiographically or is it done clinically? It is my understanding that the sacroiliac ligaments are among the strongest in the body, and for a displacement of this joint to occur a tremendous amount of trauma would be necessary.

As far as manipulation is concerned, it is all very fine and in certain instances it is beneficial. I do not agree, however, that it should be done routinely on every patient who comes along, for the simple reason that I have seen patients with low back pain and no sciatica who have gone to various practitioners not of the medical profession and following manipulation have ended up with a definite disc syndrome. Such patients, I believe, may have had some rupture of the fibers of the intervertebral disc and by manipulation that rupture was completed and the disc protruded.

As far as industrial patients are concerned, Dr. Aitken has said all there is to say about it. They are not motivated properly; they are not anxious to get well. One must evaluate them properly and surgery suould be done in those cases only as a last resort and only after psychiatric clearance. On the other hand, back surgery of a properly motivated industrial patient can be successful with ultimate satisfactory recovery.

WILLIAM R. GRANNIS (closing): I should like to take the very great liberty of amplifying one point that Dr. Haggart made. In the interest of saving time he omitted a good bit of the material which is in the written transcription.

As a temporary visitor to Dr. Haggart's department and as one who prior to going there had seen one concept of exercising for low back, I would like to say that I am tremendously impressed with the effectiveness of the simple muscle-setting exercises as carried out under Dr. Haggart's direction. He did not go into great detail about the performance of these. One point in particular is that they are not only performed while the patient stands, but also the physiotherapist teaches the patient to set the glutei and the abdominal muscles in sitting and reclining positions as well as in standing position. The objective of the exercises, at least as I understand and believe it, is to teach the patient to support his back without having to think about it, to support his back as though he were wearing a Hauser jacket or flexion brace. In other words they learn a kinesthetic sense of the position of the pelvis and are able to change their stance, which at first feels unnatural, as Dr. Haggart implied. However, it becomes natural.

Another point which Dr. Haggart stressed, which I would like to restress if I may, is that this particular adjunct of treatment should be maintained permanently after any surgery is performed. That is, the surgery is presented to the patient as only part of the treatment and he is made to realize, or so it is intended, that he must continue to observe the new stance in the use of those muscles through the remainder of his life. As the patients are seen in later visits, they are refreshed in their exercises by further visits to physiotherapy, even though in some cases it may have been two or three years since their previous visit.

HARRY E. MOCK, SR. (Chicago, Ill.): Back in 1934, two years after the disc operation was proposed at the American College of Surgeons by Naffziger, I gave my first paper against disc operations. In those two years one clinic came out with 500 cases, another with 300, others with 50 to 100, and the man who had performed only 10, 15 or 25 disc operations did not dare open his mouth in those days. I wondered how we developed this great country of ours with our dads and grandads having the same backaches as we had and having gone ahead and worked and survived and built up our country without this God-given operation of intervertebral disc.

Incidentally, I gave that paper at the state medical meeting at Rochester, Minn. That evening I got on the train with Dr. Cecil of New York and played a joke on him the next morning when we reached Chicago. We were going to the University Club for breakfast. I tipped one of the redcaps to have a chair ready to ride Dr. Cecil out to the taxicab. He kicked the chair away and when I told him it was my joke, he said, "Had you noticed my foot drop?" I hadn't. When I asked him what caused it he answered, "Oh, I had the first disc operation performed in New York. I thought if I was going to recommend it to patients, I had better go through it." He did not regret it, he said. He just regretted that he had not been able to play golf since. I have seen too many people who could not play golf after the operation.

A little over a year ago the general claim agent of one of our great railroads wrote to me and said, "We have been going over our records for a number of years, and we can't find a single herniated disc that you have done, and yet you have had over a thousand of our back cases and we have had very few lawsuits from those back cases. On the other hand, one of our best doctors has become more and more interested in operating, and he operates nearly all of our low back pains now. We have less than 10 per cent of those back to work, and we have many lawsuits. I am just wondering if you can tell me what your method of treatment is that differs so radically from his."

In the first place, we have over 3,000 people that we are working over and we have not operated upon one of them. When I suggested to my two sons that maybe dad has been wrong on this and they had better go ahead and do back operations, they both agreed that we had better continue to employ conservative treatment leaving surgery to others, so we could talk without having the temptation of operating upon some of those we would love to operate upon and maybe should. Of these 3,000 we have kept over 100 from surgery when herniated disc was diagnosed.

I want to close by telling about the Missouri farmer who brought his son to us after six months of backache. When the son was stripped, I called in my associate, Dr. John Lindquist, and said, Come here if you want to see a flat back." The farmer then said, "I am a mule buyer and no one can ever sell me a mule with a flat back, and here I didn't recognize it in my son." He took his son home. The latter carried out a set of exercises that we gave him and he got over it.

Fifty per cent of Dr. McConville's patients were between the ages of twenty and thirty-eight; the other 50 per cent after that. It is in the third decade of life that so many of these bad postural backs begin to show symptoms. I have been a victim of backaches all my life; it is the Mock tradition, sway-back. The other thing, even worse, is the long, flat back. Exercises can be adopted which will correct these, and many keep up these exercises the rest of their lives. There is need for far more conservative treatment than surgeons are

giving today. How many of those operated upon get more than two or three weeks of what they call conservative treatment before they turn to surgery? The number is pretty small in Chicago; maybe it is a great deal more here. At any rate, conservative treatment of low back pain is increasing and I welcome the fact that I have lived to see these three essayists all second the motion. It is time for a change!

B. E. McConville (closing): There was one question brought up by Dr. Giannestras in regard to sacroiliac displacement, pointing up the fact that the sacroiliac ligaments were so strong that it was doubtful a displacement was possible.

First, in regard to the question of diagnosis, of course we all have our thoughts on that and we all take a little different position on it. I have believed that a long leg on the affected side indicates a rotation of the sacroiliac joint, and due to the fact that this long leg disappears with an audible snap following manipulation, I thought it merited some consideration as to displacement.

As to the question of motion in the sacroiliac joint, if there were no motion in this joint, I doubt whether there would be an articulating surface. There are many articles written in the literature today in regard to motion of the sacroiliac joint. Several of the authors are Gray, Goldthwait, Osgood, Albee, Brooke, Cox and Sashin, all of whom have written at length about motion in the sacroiliac joint.

Regarding the question of diagnosis by x-ray, in the routine x-rays that we take I myself do not believe that I can make a diagnosis from that, with the exception of rare cases. Only occasionally, only very rarely, would I think from the x-ray standpoint that there was sacroiliac displacement by x-ray findings. However, there is an excellent article on that by Dr. Chamberlain. He has a method of positioning which I believe brings out the question of motion in the sacroiliac joint. There are others who have written at length on that namely, Ely, Stamm, Rogers and Cleaves, Mennell, Magnuson, Fisher, Radford, Pitkin and Pheasant.

In regard to the question of postural training and exercises, I did not mention it in my paper but I think it is very important and should be re-emphasized. We instruct all our patients not to turn or twist for at least one month following manipulation. We do allow straight forward bending. If they desire to move an object from one side to the other, we advise them to do so by moving their feet. We believe that if they continue this precaution for a period of a month, they are not apt to twist or bend to any great extent afterward.

The only other thing that I would like to say is that if you are considering back surgery, just ask yourself this question: If it were my back, would I want it operated upon? I think that gives you a good answer.

G. E. Haggart (closing): As stated by Dr. Ghormley, diagnosis is the first requirement. The significant details of what we carry out on that score will appear in the printed report. Then follows a decision on treatment. As I have stated, we believe the majority of patients with low back and/or sciatic pain should be handled on a conservative basis.

As to treatment by manipulation, I take issue with Dr. Wagner if I understood him correctly to say that he believes such therapy can be carried out before studies are made as to whether there is an immediate extradural or intradural pathologic condition. I believe this is a mistake, as is well illustrated by a patient on whom we made a clinical diagnosis of intervertebral disc protrusion and started on conservative treatment. He did not improve and a pantopaque myelogram was performed. Fluoroscopic examination and roentgenograms were clearly indicative of disc protrusion and therefore operation was advised. The patient decided that he did not wish surgery and consequently was urged to carry on with the exercises and report if his symptoms became worse. Instead he went elsewhere and his back was manipulated. I do not know the technic employed. Some forty-eight hours later he appeared in my office with a complete foot drop. He was immediately hospitalized and operated upon after appropriate preliminary preparation. A large, completely extruded fragment of degenerated disc tissue was removed from the fourth lumbar space. Although the patient no longer experiences incapacitating sciatic pain, he has not obtained complete recovery from the foot drop eighteen months after operation.

Manipulation of the back has a definite place in the treatment of these patients but I wish to emphasize my belief that such treatment should be performed only after a complete study, including a myelogram.

I am in complete accord with the remarks by Dr. Aitken based on these cases obtained from the files of a large compensation carrier. He noted that as long as compensation is paid, these cases can be followed up, which represents a very real advantage over the individual investigator. In particular do I agree with his emphasis on the importance of adequate conservative treatment not only as such but also as a diagnostic aid in the management of these patients.

# MALIGNANT SYNOVIOMA OF THE ANKLE

DONALD C. CONZETT, M.D.
*Dubuque, Iowa*

VARIED opinions have been expressed concerning the role of trauma in the etiology of synovioma. The following case is reported to add support to the theory that injury is an inciting cause in the production of this tumor.

R. B., an eleven year old boy, on January 23, 1952, sustained an injury to his right ankle while playing basketball. He was seen by his family physician a few hours later and because of the pain, tenderness and characteristic swelling adjacent to the external malleolus, the injury was treated as a sprain using an adhesive boot. With this support the lad continued his school work. Three weeks later he was again seen and, although the pain had lessened, the swelling remained. Again an adhesive support was applied. A week later, when the swelling had not subsided, he was referred elsewhere and an x-ray taken of foot and ankle. Following a negative report the patient was advised that the injury was a chronic sprain and alternating hot and cold applications were suggested. A month later on April 5th pain suddenly developed over the lateral aspect of the ankle joint and a noticeable discoloration, mottled in appearance, was evident over the persistently swollen submalleolar area. A week later the patient came under our care. Examination revealed an otherwise healthy boy with a multilobular mass, the size of a golf ball, just distal and anterior to the lateral malleolus. There was only moderate tenderness to palpation and the impression was that the tumor was of cystic consistency. It was slightly movable and not attached to the malleolus or skin. Ankle and foot motions were entirely normal. A further x-ray was ordered and extensive calcaneal destruction noted. The original films were available and restudy with the knowledge gained from the second examination showed beginning bone involvement at that time. An immediate biopsy was done and frozen sections made. Two days later, after advising the parents of the gravity of the situation, an upper thigh amputation was done. A careful dissection of the limb by the pathologist disclosed no involvement of lymph nodes. A chest film postoperatively was negative. The stump healed well and the boy has since been fitted with a suction socket prosthesis. When seen recently, five months postoperatively, he was in apparent good health, had gained thirty pounds, and was attending school. Despite this, however, a chest film on September 13, 1952, revealed a 3 cm. nodular metastasis at the base of the right lung and 2 cm. metastic nodule at the left base. Lungs were otherwise clear, and the heart, diaphragms and pleura normal. The prognosis is an unhappy one.

As defined by Pack, synovial sarcoma represents those malignant neoplasms which arise from synovial tissue of joints, tendon sheaths and bursae and which present histologic features characteristic of synovial tissue. Although this entity was first described by Stuer in 1893, Smith in 1927 originated the term of synovioma. Considerable confusion arose over the histopathology in that some authors ascribed the tumor to be of epithelial origin; others of endothelial origin and still others that it was a specially differentiated type of mesodermal cell. The last-named now seems to have greatest support. There seems to be no established gross pattern, for Pack and Irving comment on the marked variation encountered in that the tumor may be encapsulated, circumscribed, or diffuse-infiltrating, surrounding structures and invading bone. It may incorporate adjacent tendons and frequently it is impossible to determine the exact site of origin. Such was the condition in the case reported. Briggs mentioned that in his nine cases all had a tendency to cyst formation, were poorly encapsulated, usually lobulated, and soft in consistency. In their report of sixty cases Pack and Irving noted no sex preponderance although it seemed to be a disease of young adults, the average being 36.3 years. The location of the tumor in their series was knee 21.7 per cent; foot 18.3 per cent; hand 16.7 per cent, and other joints in smaller percentages.

Metastasis usually occurs by blood stream and in one large series 65 per cent showed metastatic deposits in the lungs, occasionally in the regional lymph glands and 8 per cent in bone. Other tumors must be differentiated. Chondromas and osteomas are of harder consistency and attached to bone. Lipomas are softer and radioluscent. Xanthomas are fre-

quently difficult to differentiate but their site is usually characteristic, occurring on flexor tendons of fingers or wrist. Carcinomas affect the overlying skin.

Treatment is likewise controversial. It is generally agreed that mutilating biopsy is hazardous and that local excision is well nigh impossible. Amputation early and at a high level seems to offer the greatest hope of survival. The effect of postoperative radiation has questioned value. Pack and Irving state that radical surgery is the one suitable method of eradicating synovial sarcoma and that radiation therapy may be used to supplement surgery, or for palliation. On the other hand, Haagenson and Stout state that radiation is valueless. Jonsson of Stockholm believes the tumor to be radio sensitive. Fisher, reviewing forty-three cases from the foreign literature, comments that most authors believe the tumor to be radio resistant. He then makes a thought-provoking statement when he questions whether the tumor may not be activated into malignant growth by operative intervention and cites these two facts: "No untreated patient, with the diagnosis first made by autopsy has yet been reported and no reported patient has developed metastasis until after operation."

The prognosis of this entity is a grim one. In Pack's series there was a recurrence rate of 63.3 per cent but interestingly the survival rate is higher in the younger age group. No patient over fifty survived five years. It must be remembered that many of these reported cases had early attempts at local excision, some at a considerable time prior to definitive surgery.

The role of trauma is inconclusive. In Pack's series nine of the sixty cases gave a definite history and in five of these the tumor appeared immediately or shortly after trauma. To this author this strongly suggested that trauma merely called attention to a pre-existing lesion or that possibly hemorrhage occurred within the tumor thereby increasing its size. Briggs' series had an history of trauma in 33 per cent, but the earliest tumor development was three months following injury. Lazaries and Marks mention 25 per cent of their cases having a history of trauma but they believe, as do Haagensen and Stout, that it is of little significance. In an allied series of 327 xanthomas studied by Galloway, Broders and Ghormley they found an etiologic incidence of trauma in 19 per cent.



SUMMARY

A case of synovioma of the ankle in an eleven year old boy is reported with a short review of recent reports. Although definite trauma preceded the appearance of the tumor mass, it can be only presumptive evidence that injury initiated the growth. Further recording of cases is suggested and the hope expressed that early diagnosis may be made so that definitive and radical treatment can be instituted.

REFERENCES

Black, W. C. Synovioma (synovialoma) of foot; case. *Am. J. Cancer*, 39: 199–206, 1940.

Bennett, G. A. Malignant neoplasms (synoviomata); Study of 32 specimens registered at Army Institute of Pathology during wartime period 1941–1945. *J. Bone & Joint Surg.*, 29: 259–291, 1947.

Bedrick, J. J. and Zawadski, S. A. Malignant synovioma; case. *Mil. Surgeon*, 97: 374–377, 1945.

Maretz, W. H. Malignant tumors arising from membranes with report of cases. *Surg., Gynec. & Obst.*, 79: 125–132, 1944.

Stanford, S. and Harne, E. A. Malignant synovioma. *J. Bone & Joint Surg.*, 25: 883–891, 1943.

Fisher, H. L. Sarcomesothelioma (sarcoendothelioma or synovioma). *Am. J. Pathology*, 18: 529–553, 1942.

Briggs, C. D. Malignant tumors of synovial origin. *Ann. Surg.*, 115: 413–426, 1942.

De Santo, D. A., Tennant, R. and Rasahn, P. D. Synovial sarcomas in joints, bursae and tendon sheaths; clinical and pathologic study of 16 cases. *Surg., Gynec. & Obst.*, 72: 951–981, 1941.

Harris, V. C. J. Three cases of synovioma. *Brit. M. J.*, 1: 447–448, 1948.

Stewart, M. J. Benign giant cell synovioma. *J. Bone & Joint Surg.*, 30-B: 522–527, 1948.

Myerding, H. W. and Flachman, F. L. Tumors of bone and of synovial membrane; progress in orthopedic surgery for 1946. *Arch. Surg.*, 57: 871–896, 1948.

Wright, C. J. E. Rapid recurrence of giant-cell synovioma of tendon sheath. *J. Path. & Bact.*, 61: 271–274, 1949.

Liu, Y. Malignant synovioma; 2 cases. *Chinese M. J.*, 67: 238–242, 1949.

Pack, G. T. and Ariel, I. M. Sarcoma (malignant synovioma) report of 60 cases. *Surgery*, 28: 1047–1084, 1950.

Galloway, J. D. B., Broders, A. C. and Ghormley, R. K. Xanthoma of tendon sheath and synovial membranes; clinical and pathologic study. *Arch. Surg.*, 40: 485–538, 1940.

Meyerding, H. W. et al. Tumors of bone and of synovial membrane. *Arch. Surg.*, 60: 170–181, 1950.

King, E. S. J. Tissue differentiation in malignant synovial tumours. *J. Bone & Joint Surg.*, 34B: 97–115, 1952.

# TRAUMA AND DUPUYTREN'S CONTRACTURE*

JOHN J. MOORHEAD, M.D.
*New York, New York*

THE alleged relationship of trauma to contracture of the palmar fascia has been in dispute since 1832 when the Baron Guillaume Dupuytren[10] of Paris proclaimed the exact site of this sclerotic process. His first operative case was a wine merchant who gave a history of lifting a cask in 1811 and noted a crackling sensation in the palm of the hand. However, he paid no attention to this incident until contractures of his fingers became annoyingly noticeable and disabling. It is interesting to read Dupuytren's remarks during the operation he performed twenty years later (June, 1831) as recorded by Paillard and Marx[28] and his description of this still unsolved pathologic process is as applicable today as it was a century and two decades ago. It is notable that this operation was performed without any inhalant anesthesia because ether was not used in France until 1848. Dupuytren's second operation (December, 1831) was the case of a coachman who was supposedly afflicted because his steeds required him to use both hands to control their antics. Hence this is the essential historic basis for the alleged responsibility of trauma as the causative factor, and this viewpoint is still maintained by some proponents, although it is generally conceded by more experienced observers that traumatic origin is not the etiologic factor. The present authoritative trend is to agree that an initial or single trauma plays no part; however others continue to assert that the relationship, if any, is due to repeated, persistent or occupational causes incidental to use of the hand or hands. Here then briefly is the background of a process allegedly due to trauma which has assumed certain medicolegal stature on the theory of aggravation or acceleration on the basis of occupation in which manual labor is an outstanding factor. Such a viewpoint in my opinion is unjustified for a variety of reasons.

This condition attracted my attention forty-three years ago when I read and published an article[1] with the same title used today. The endeavor at that time was to show that a study of several hundred street railway employees failed to indicate any traumatic relationship, even though most of those examined were beyond their fourth decade and had been for years in occupations requiring constant use of their palms. That same article with few modifications has been used by me in the three traumatic surgery textbooks subsequently published[1] by Saunders in 1917, 1931 and 1945.

Dupuytren's contracture may be said to be a chronic, painless, self-progressive sclerosis of the palmar fascia assumed to be due to a variety of causes, none of which are actually responsible. Of all the alleged factors based on neoplastic, neurologic, endocrine, traumatic, occupational, infective, gouty or rheumatic sources the only speculation concededly related is heredity. It is a curious paradox that after almost a century and one-third no etiologic factors are agreed upon, and that Dupuytren's original uncorroborated observation continues to reflect a certain type of surgical thinking. Thus we are reminded that all of us are swayed by the repetition of what somebody else is said to have said.

This reliance upon Dupuytren's original report is the more remarkable because Felix Plater in 1614 and Sir Astley Cooper in 1818 antedated Dupuytren's initial publication. Of these Cooper alone accused the palmar fascia as the site of this lesion and affirmed that the adjacent joints, tendons and soft parts were not implicated. It is noteworthy that competent writers on this subject refer to the relentless but painless leisurely progression of the process; the tendency for remission and accession; the natural desire of the patient to name a traumatic incident as the source; and the added observation that little if any attention causes comment until ridges or contractures appear. Curiously enough two recent observations confirm the clinical fact that the condition may be well advanced among our own friends without our

* From the Department of Surgery, New York University Post-Graduate Medical School, New York, N. Y.

knowledge. As proof of this let me say that a surgeon known to many of you, and a former chief of mine with whom I have been associated for decades, recently showed me both nodulated palms and said he had known of this condition for years, and that two of his brothers had the same process in a more advanced stage. The same experience was duplicated in a clergyman who has a bilateral second stage condition and hitherto believed for years that he was susceptible to callosities.

Another peculiar baffling feature is that this condition with the greatest precision and regularity registers on the ulnar border of the hand near the base of the ring and little fingers. Oddly enough this is the segment of the palm least concerned with manual effort and the area most protectively folded upon itself in grasping and other motions. In the usual use of the hand the thumb, index and middle fingers most often participate and yet they are least involved in this puzzling etiologic problem that is so clinically apparent on mere inspection and palpation. This fibrous apron called the palmar fascia has a counterpart in the plantar fascia; and although the latter is almost constantly subjected to pressure and friction, it is very rarely the site of this lesion. Likewise the dorsum of the penis has a fibrous area that occasionally is involved and is known as induratio penis plastica, or Peyronie's disease. A third exceptional site is over the so-called knuckle pads at the middle dorsal interphalangeal joints. Thus clinically and pathologically there are four areas in which this process may appear but the diagnosis is very rarely made unless the palmar area is involved. Other related observations are that this disease is usually unrecognizable until after the fourth decade, although there are several recorded cases appearing in earlier adult life and a few cases in children. A most exceptional instance is that of male identical twins reported by Harold Couch,[8] of Toronto University, who kindly wrote me[9] that his patients were farmers in their sixties, and the process was unilateral involving and progressing in identical fingers to the same degree. This case is biologic proof that might be questioned if fraternal and not identical twins were involved. It is conceded that the condition appears in the sclerotic period of life and that it has periods of accession and remission that may be ascribed to various sources actually unrelated. Thus palmar fasciitis is another confirmation of our credulity in assigning an unknown disease to a definite incident in the few cases in which we may be interested.

Trauma is held to be a factor in a great many conditions wherein the actual origin is unknown or speculative. This recalls that in my student days we were taught to state that alcohol, syphilis, gout and trauma were the factors in many diseases of unproven ancestry. The "and trauma" is still an uncorroborated culprit, just as if trauma had not responsibilities enough. Curiously enough this association is urged in the most diverse conditions clinically and pathologically, notably in neoplasms benign and malign. There is a refuge in this class of uncertainty that has helped me throughout the years, and it is the self-propounded question, "What usually and ordinarily happens under a given set of circumstances?" If we apply that precept to this topic, what is the answer in respect to the relationship of trauma? In trauma to the hand, single, multiple, repeated, superficial, deep, recent or remote, do you find any sustained causative relationship with this process? If this sclerotic process is the result of an inflammatory agency, as is asserted in a few reported cases, there is a conflict in that line of reasoning because most of us are agreed that any inflammatory process in the hand, especially fascial, is exceedingly painful and registers unmistakably by the ancient signs of inflammation such as redness, heat, pain, swelling and interference with function. Further, let us never forget another clinical aphorism, namely, that there is no incubation period in trauma and no long delay in declaring itself. There is no accounting in these highly speculative opinions for the relative absence of this condition in women, in Negroes and in many occupations demanding daily use of the palms. I am impressed by the observation that chauffers and motorists generally are singularly free from this lesion even though by my own tests the ulnar borders of the hand are subjected to pressure each time the steering wheel is grasped or the hand brake operated. Likewise drillers, road builders, laborers, farmers, or gardeners, and others using wooden or metal tools with cylindrical handles should be affected. If it is due to trauma, single, repeated or occupational, it is surprising that manifestations of the disease are so relatively rare, affecting

only between 1 or 2 per cent of the population. Why is it not a clinical problem encountered by a group so trauma-minded as our own organization if occupation or any other external agency in this mechanistic age is a producing or aggravating factor?

Within the past several years I have examined 142 cases in which compensation claims have been made, in the main by a group of employees in the brewing industry. The assertion was that their occupation required frequent use of the hands in their work with tools and apparatus, barrels, wooden and cardboard containers, and in the driving and loading and unloading of trucks. A few of these claimants asserted that a definite accidental wound or traumatic incident started the process years ago. Nearly all however said their attention was drawn to the condition by a survey made at the suggestion of their employee organization. Few of those examined had any pre-employment survey. In no instance was the date of onset precisely known, and only a very small minority had sought treatment or paid heed until the condition caused considerable nodulation or contracture. The pertinent statistics in this group of 142 indicate that practically all of them were over forty; that they were unable to fix the date of onset; that there was no special occupational factor requiring steady use of the palms; that many of them wore gloves at work; that in more than half the condition affected the right hand in the right-handed; the left hand was alone involved in a lesser degree, and there was bilateral involvement in about three-fourths; that the predominant site was at the base of the ring or little finger (or both); that there was no neural or vascular involvement; that the plantar area was also involved in six cases; that there was only one case in which the knuckle pads were involved. The significant facts in this selected group again indicate that this disease is slowly and painlessly progressive and that the possessor becomes aware of his condition only when his attention is called to it by apparent nodulations and contractures, or as the outcome of examination. It is noteworthy that such obvious and perhaps disabling deformities may exist and yet the possessor has acquired a tolerant immunity because of years of adaptation. Most of these 142 men referred to the typical nodulations as callosities, and the contractures as signs of arthritis.

In an attempt to cull the best available recent literature the library of the American College of Surgeons sent me twenty-five reprints. One of the most interesting and informative was by C. E. Corlette,[7] of Sydney, Australia, who writes from a long surgical experience, and incidentally has this lesion himself and refers to others in our profession who have this disease unknown to their friends and associates. He is convinced that heredity is practically the sole cause. From another source I have read a most praiseworthy encyclopedic presentation on the subject by Tord Skoog[36] of Upsala, Sweden, who served as a British Council Scholar in London with the well known Sir Archibald McIndoe. Sir Archibald has a special operative technic similar to Bunnell's and Skoog uses as his background forty of his chief's operative cases and adds ten of his own. These fifty operations gave him the impetus to survey the recorded worldwide field in a monograph of 190 pages. His bibliography lists 377 references and he discusses in detail the etiologic factors, and finally arrives at a conclusion very similar to that stated by Robert Abbe,[1] then the well known professor and surgeon of New York who wrote me his opinion in the article I published long ago. It is informative to learn from Skoog that of 207 epileptics examined in Sweden 42 per cent had palmar, 26 per cent knuckle pad and 8 per cent plantar involvement. This association with epilepsy had not hitherto been especially stressed in the publications in our own country. A series as large as this again shows that a statistical sampling of a special group may have implications of undue importance unless balanced against controls for comparison. It is very interesting to note that after a most exhaustive search of the literature, a study of well documented cases, and even the employment of the electronic microscope, Skoog evolves a theory of origin that adds to the confusion of this riddle and mocks a diligence on his part that willingly arouses our envy and our pride. Sterling Bunnell[6] in his outstanding textbook and a wealth of experience, expresses the opinion that corresponds with seasoned informed judgment when he writes: "There is often a desire to claim that a Dupuytren's contracture is due to trauma from labor, and early writers held this view. Though it is the usual and natural assumption of a patient to ascribe the condition to the effect of a

traumatism it is clear nevertheless that the cause of the condition is unrelated to trauma." It is asserted or admitted by some that this lesion is a physical endowment and that those who have it conferred by heredity are susceptible and thus develop it when others do not under identical circumstances. I daresay this line of reasoning would give the same answer for cerebral palsy, multiple sclerosis and others of undetermined origin. This belief is but another of the many surmises that surround this etiologic riddle that has been a puzzle since it came into prominence in France 121 years ago.

No one has yet told us why this lesion continues to register in one location throughout the years as if a branding iron had trade-marked the recipient only on the ulnar border of the hand. There are fascial fields a plenty and yet these escape; and the attack is less often upon the no-collar than upon the white-collar worker; and the process with great regularity does not show on the hand until after the fortieth year; and also six to one it affects men although women do plenty of manual work. Manual workers are supposedly more prone, yet the best-informed say the proportion is approximately fifty-five non-manual to forty-five manual. All agree that the ring finger area is the site most often affected, the little finger next, and then the middle finger follows, and least often the index digit and the thumb. Here then is another paradox in that the least used part of the hand gets the brunt of the pathologic storm and the actively functionating area escapes. Then we also refer to the statistical evidence given by Skoog's analysis of 2,278 cases quoted from a selection of twenty-eight writers in sixty-seven years (1881–1948). These figures show the right hand affected in 663 (29.1 per cent), the left hand in 364 (16.0 per cent), and bimanually in 1,251 (54.9 per cent). It is to be remembered that these statistics, like others on diverse subjects, are the collected totals of writers from many parts of the world and that there has been no universal or standard method of assessing or registering the findings. There is, for example, no way to reconcile the large percentage of bilateral involvement unless we recall that it is a well known clinical fact that usually the lesion begins in one hand and later appears in the other also.

The grading or assessment of the extent of involvement has been attempted by many authors. For my own purposes four degrees of involvement are listed as follows: *First degree:* 1° = nodulation, ± dimpling of the palm. *Second degree:* 2° = bands or rugae, ± nodulation. *Third degree:* 3° = contractures of finger, ± bands. *Fourth degree:* 4° = combinations of 1°, 2°, 3°. The duration of each degree or stage is difficult to estimate, but from a clinical standpoint a period of five years may be said to elapse between each phase. Thus a patient with nodulation (1°) and bands (2°) may be said to have had the process between five and ten years. Between the third and fourth degree there is usually a duration period between fifteen and twenty years.

There are instances in which the progression is slower, faster or stationary; however the general advancing nature of the lesion is one of the main features. It is exceedingly rare in my experience to find a person who knows when his ailment first appeared, and this "don't know" or "take it for granted" phase is another peculiarity. It is paradoxic to reflect that a visibly deforming and disabling disease can go unrecognized in irreplacable segments of the body only less important than the eyes. It is also odd that manual workers and others will remain complacent for years with so apparent an ailment as this palmar fascia fibrosis. There is one other feature worth mentioning, namely, that the palmar fascia is not as subcutaneous as many believe. The skin and a substantial layer of fatty tissue are superimposed to aid in promoting two main functions of this fascia, namely, to keep the tendons from displacement, and to provide a movable elastic structure permitting mobility of the palm. Incidentally if repeated impacts on the palm caused or promoted this fibrositis, it must be a very selective action derived from an external source that does not first register at the site of friction or impact. It is hard to believe how external irritation can evade the more superficial skin and yet affect the deeper and tougher fascia, a type of contra-coup it might be called. I am also impressed by the lack of callosities over the nodules and ridged bands in even advanced cases and am unable to understand how any external traumatic agency can exert so selective a role. It reminds me of the old days when a blow on the abdomen was the asserted cause of traumatic appendicitis. Again the culprit trauma bore the brunt of responsibility, and

indeed to this day has been accused of almost everything except tonsillitis from tight collars, and an enlarged prostate from a sit-down strike.

*Diagnosis* is usually easy because the nodules and the rigid striae are pathognomonic, and the finger contractures imitate only tendinous involvement usually due to definite injury, infection, arthritic or birth sources. It is helpful in the diagnosis to extend the affected digits and note how the involved fascial bands thus stand out more prominently.

*Treatment* to be effective should be in the early stages, and complete excision of the entire fascia is the best guarantee against recurrence. A wide variety of exposures are described, all of which aim to uncover the lesion and avoid damage to the neural, vascular and tendinous adjacent anatomy. The skin may be so devitalized that autogenous grafting becomes necessary.

In this attempt to prove that trauma and Dupuytren's contracture are unrelated I have only one reservation and that is the unknown effect a fascial space infection would have on an already developed case of this disease. This is a highly speculative surmise and it is mentioned only to indicate that such a combination has not been encountered in my reading or personal experience.

If asked for the reasons why Dupuytren's contracture is not caused by injury nor aggravated nor accelerated by external irritation or occupation, the following summation is offered: (1) This is an ancient disease of unknown origin that has been ascribed to a wide variety of assumed sources, none of which except heredity are conceded by the best-informed. Familial origin is traceable often in 40 per cent of the cases. (2) It is relatively rare, occurring in only 1 or 2 per cent of the population; if due to external sources it should be very prevalent and occur frequently in hand workers. Statistics indicate that it appears somewhat more often in the white-collar than in the no-collar group. It is rare in women even though many of them are manual workers and users of tools; also rare in Negroes and some other races. (3) Usually it appears only at an age when sclerotic changes are manifest elsewhere. It is inevitably progressive having stationary, as well as periods of accession and remission. (4) It begins in one hand, and often later appears in the other hand. Date of onset is rarely known because it is painless and not disabling. (5) With great regularity it registers first with a nodulation at the base of the ring and little finger. Practically never affects the thumb and index fingers which bear the brunt of manual activity. (6) It exists a long time unknown to or unobserved by the individual or associates because it is painless and usually slowly progressive with periods of accession and remission. This insensitivity is remarkable because the area affected is notoriously hypersensitive, notably to inflammatory irritants. (7) Operative exposure demonstrates a wide spread sclerotic process, suggesting intrinsic rather than extrinsic origin. The skin is seen to be affected by progression from the subjacent fascia, and is not the site of the originating focus. (8) The plantar fascia and the palmar fascia have a similar structure and function, yet sclerosis in the sole is very rare despite repeated impact and friction. How to account for the pathologically identical knuckle involvement adds to the non-traumatic proof. (9) It appears at an age, usually after forty, when sclerotic processes are evident elsewhere. (10) It occurs in either hand and may involve the right hand of the right-handed, or vice versa, and often it is bilateral and quite symmetrical. (11) In clinical experience it is very rare considering the number of injuries, infections and other lesions involving the hands, and it never occurs as a complication even after fascial involvement. (12) The clinical observation that the site, symptoms and treatment are agreed upon by the experienced, suggests that the etiology should also be uncontroverted if trauma or any other source was responsible. Thus the conclusion seems warranted that this is a *disease* of unknown origin and not a *traumatism*.

REFERENCES

1. Abbe, R. Dupuytren's finger contraction. Further remarks on the theory of its nervous origin. *M. Rec.*, 33: 237, 1888.
2. Adams, H. D. Dupuytren's contracture. *S. Clin. North America*, 22: 899–906, 1942.
3. Ayre, W. B. Dupuytren's contracture. *Canad. M. A. J.*, 54: 158–160, 1946.
4. Baxter, H. et al. Cortisone therapy in Dupuytren's contracture. *Plas. & Recon. Surg.*, 9: 261–273, 1952.
5. Bode, E. Dupuytren's contracture. *Beitr. z. klin. Chir.*, 179: 1–160, 1949.
6. Bunnell, S. Dupuytren's Contracture. Surgery of the Hand. Philadelphia, 1944, 1948. J. B. Lippincott Company.

7. Corlette, C. E. Dupuytren's contracture. *M. J. Australia*, 2: 178–182, 1944.
8. Couch, H. Identical Dupuytren's contracture in identical twins. *Canad. M. A. J.*, 78: 225, 1938.
9. Couch, H. Personal communication. March, 6, 1952.
10. Dupuytren, G. De la rétraction des doigts par suite d'une affection de l'aponéurose palmaire. Description de la maladie. Opération chirurgicale qui convient dans de cas. *J. univ. et hebd. de méd. et de chir. prat.*, Paris, 2s. 5: 353–365, 1831. Also: *Lancet*, 2: 222–225, 1834. Also: trans. by L. Clark: London, New Sydenham Soc., 1854. Also: *M. Classics*, 4: 127–150, 1939.
11. Flandin, C. et al. Treatment of palmar aponeurosis and tendinous retractions. *J. A. M. A.*, 144: 858, 1950.
12. Gray, G. A. Dupuytren's contracture. *U. S. Armed Forces M. J.*, 1: 49–57, 1950.
13. Greenberg, L. Dupuytren's contracture of palmar and plantar fasciae. *J. Bone & Joint Surg.*, 21: 785–788, 1939.
14. Hammond, G. and Dotter, W. E. Quadrilateral Dupuytren's contracture. *Guthrie Clin. Bull.*, 18: 34–38, 1948.
15. Jones, A. R. Dupuytren's contracture. *M. Press*, 206: 14–16, 1941.
16. Editorial. Dupuytren's contracture. *J. A. M. A.*, 137: 1177–1178, 1948.
17. Kanavel, A. B., Koch, S. L. and Mason, M. L. Dupuytren's contraction. *Surg., Gynec. & Obst.*, 48: 145, 1929.
18. Kaplan, E. B. Pathology and operative correction of finger deformities due to injuries and contractures of the extensor digitorum tendon. *Surgery*, 6: 35–47, 1939.
19. Kaplan, E. B. The palmar fascia in connection with Dupuytren's contracture. *Surgery*, 4: 415–422, 1938.
20. Kehl, K. C. Dupuytren's contracture as a sequel to coronary artery disease and myocardial infarction. *Ann. Int. Med.*, 19: 213–223, 1943.
21. Koch, S. L. Acquired contractures of the hand. *Am. J. Surg.*, 9: 413–423, 1930.
22. Koch, S. L. Dupuytren's contraction. *J. A. M. A.*, 100: 878–880, 1933.
23. Meyerding, H. W. The treatment of Dupuytren's contracture. *Am. J. Surg.*, 49: 94–103, 1940.
24. Meyerding, H. W. Bilateral Dupuytren's contracture. *Proc. Staff Meet., Mayo Clin.*, 10: 801–804, 1935.
20. Meyerding, H. W. Dupuytren's contracture. *Arch. Surg.*, 32: 320–333, 1936.
26. Meyerding, H. W. et al. The etiology and pathology of Dupuytren's contracture. *Surg., Gynec. & Obst.*, 72: 582–590, 1941.
27. Moorhead, J. J. Dupuytren's Contracture, Traumatic Surgery, 1917, 1921, 1923 (Saunders); Traumatotherapy: Treatment of the Injured, 1931 (Saunders); Clinical Traumatic Surgery, 1945 (Saunders); Injury & Dupuytren's contraction, *Post-Grad. M. J.*, Aug. 1909; Med. & Surg. Reports of Bellevue & Allied Hosps., vol. IV, 1909–1910.
28. Paillard and Marx. Notes of the Surgical Clinic at the Hôtel Dieu, Paris, Dec. 8, 1831.
29. Palmer, L. A. Bridge operation for Dupuytren's contracture. *Am. J. Surg.*, 68: 351–354, 1945.
30. Parsons, A. R. Dupuytren's contracture. Treatment by massive doses of vitamin E. *Irish J. M. Sc.*, 272–274, 1948.
31. Powers, H. Dupuytren's contracture one hundred years after Dupuytren. *J. Nerv. & Ment. Dis.*, 80: 386–409, 1934.
32. Reichel, P. Trophic nerves and Dupuytren's contracture. *Chirurg, Würzburg*, pp. 97–144, 1946.
33. Ritchey, S. J. The use of cortisone in Dupuytren's contracture. *U. S. Armed Forces M. J.*, 3: 811–818, 1952.
34. Ross, J. and Annan, J. H. Dupuytren's contracture; a clinical review. *Ann. Surg.*, 134: 186, 1951.
35. Skinner, H. L. Dupuytren's contracture. *Surgery*, 10: 313–317, 1941.
36. Skoog, T. Dupuytren's contracture. *Acta chir. Scandinav.*, 96: 1–190, 1948.
37. Stern, E. L. An operative procedure for Dupuytren's contracture. *Am. J. Surg.*, 54: 711–715, 1941.

DISCUSSION

Laurie H. McKim (Montreal, Quebec, Can.): In regard to Dr. Moorhead's paper I was particularly interested in those points dealing with the historical aspects of the condition and the original descriptions. I am very sorry, however, that space did not permit him to take up one part of the affair in which I am greatly interested, and that is—I am going to say something that I am sure will not be agreeable to a great many of the members—the matter of treatment by x-ray.

For a number of years, approximately twenty, in the Montreal General Hospital, Dr. J. W. McKay and I have followed this thing rather carefully. In our experience, which has been rather wide, we have found that in properly selected cases of the early formation of this condition, the thing can be stopped dead by x-ray treatment. Dr. McKay emphasizes fractionated dosage slightly under the erythema skin dose, and the dosage usually comes to approximately 800 r.

I am not claiming that this is going to cure an old case of Dupuytren's contracture where the finger is down in the palm or anything like that. I am referring to the acute case in a man who has possibly had a slight nodular condition and the thing suddenly starts to grow. If we examine the palmar fascia removed in those cases, we find fibroblasts are present in quantity, and we know that fibroblasts are sensitive to x-ray. We use x-ray treatment for keloid, and we know it is of use only in the early stages of keloid, not when there is a great, broad scar.

I wish to go on record in behalf of Dr. McKay and myself as to the successes we have had with this method. I do not know how many cases there are, but we have had a good many. The only regret

we have is that we did not start twenty years ago keeping pictures of these cases, which we have not done, but we have a great many of them alive and going around. Some of them had begun to get an actual slight contracture, the finger coming down slightly. All of these were greatly improved, the fibrous nodules disappeared and the finger once more became straight.

MICHAEL LIVINGOOD MASON (Chicago, Ill.): I was very much interested to hear Dr. Moorhead's discussion of the etiology of Dupuytren's contracture because his ideas are pretty much the same as ours. I do not think anyone has actually proved that trauma has anything to do with it. There is a possibility that trauma may be the thing that sets off the mechanism which must already be built into the hand, and that mechanism is in all probability an inherited mechanism. We do not find a history of heredity in every case. I suppose one of every three will give such a history. Often we will have a patient with Dupuytren's contracture and when we ask him about his father and grandfather he has no knowledge concerning the presence of this disease within the family. The next time he comes in he says, "I have found out now that my father had this," or "that my uncle had this." The increase in the heredity history as the patients become aware of that possibility is quite remarkable.

There is one thing that has interested us in the last few years in Dupuytren's contracture which I cannot explain at all, and that is that there seems to be some tendency for the association of this disease with epilepsy. This never struck me until I operated upon a boy of nineteen or twenty, an epileptic, with Dupuytren's contracture. We talked with some of our neurologists who were handling large epileptic clinics, and one of our associates, Dr. Bell, and this chap are investigating that association. They are finding, much to our surprise, a fact which we cannot explain, a rather high percentage of epileptics with Dupuytren's contracture.

JOHN J. MOORHEAD (closing): I was very much interested in what Dr. McKim said with regard to the use of x-ray. In my paper I made mention of the use of x-ray. In some cases it seems to have a favorable effect. Whether it happens to hit those cases that are in a quiescent period or stationary period is unknown. Personally, I have not had very much success with the use of x-ray.

In regard to heredity it is awfully hard to get a family history in practically every population, especially the type of people that we have in New York City who come from all over the earth.

As to the relationship of epilepsy, in the article that I referred to and briefly mentioned, by Skoog of Sweden, he talks about a group of people that he examined in which there was a very large number of epileptics. I state that percentage in my paper, and I also say that in our own country that relationship has not been verified. I hope the day will come when this organization (as I recommended some years ago when I read a paper on so-called traumatic hernia before you) will take a stand and classify things which are definitely traumatic, which are not traumatic, and which may be traumatic—a bureau of standards, so to speak, in reference to traumatism—because you represent throughout this country, Canada, and elsewhere, the largest group of men who are interested solely or nearly solely in this disputed problem of the relationship of trauma to injury.

# COMPARATIVE EXPERIMENTAL STUDIES ON THE TREATMENT OF TRAUMATIC SHOCK*

CHARLES L. FOX, JR., M.D., WALTER L. MERSHEIMER, M.D., SIGMUND LASKER, M.S. AND JAMES M. WINFIELD, M.D.

*New York, New York*

THE problem of evaluating preparations now available for the treatment of shock following extensive burns has been approached in two ways. One was to produce standardized burns in laboratory animals and gauge the efficacy of a number of solutions by determining their effects on mortality. The second was to conduct comparative studies in patients with extensive burns.

In the past many investigators have used dogs as the experimental subject. Experience in our own and other laboratories has shown that dogs are unsatisfactory subjects for several reasons: Dogs differ greatly in age, weight, etc., and it is difficult technically to use enough animals to obtain statistically significant results. After extensive burns dogs do not go into shock as man does. This may be related to the well known difference in blood chemistry between man and dog, namely, the dog red cells contain sodium salts but practically no potassium while the red cells of man contain a great deal of potassium. For these reasons most of our experiments were performed on mice; their blood is similar to that of man, after being burned they go into severe and protracted shock similar to man, and finally, it is possible to use large groups of animals of uniform age and weight to test several therapeutic agents on the same day under the same conditions.

In all our experiments the mice were lightly anesthetized with ether and then burned by immersion to the axilla in water at 66°C. for six, seven or eight seconds. An automatic electric timer was used so that all mice were immersed for an identical period. The area of burn represented 60 per cent of body surface. The mice were then divided into groups and therapy begun within one hour and concluded within five hours. No therapy was given on subsequent days. Measurements of blood pressure, blood volume or hematocrit or other determinations were not considered in evaluating these agents. The sole criterion was whether the animals lived or died in comparison with a group of animals receiving no therapy whatsoever.

It soon became apparent that slight modification in the period of scalding altered the survival of the non-treated mice from about 50 to 100 per cent. Thus in the tables which will summarize our therapeutic experiments it is important to observe the mortality in the group of untreated mice. In general, a less prolonged burn caused less than 100 per cent mortality in untreated mice, and in these instances some of the therapeutic agents appeared to be quite effective.

In the tables the figures represent the per cent of mortality in each group for the first three days after scalding. The number of animals in each group is also given. In view of the fact that there were differences in the sodium content of the various colloid preparations tested, all mice received additional amounts of solution of sodium salts so that the total amount of sodium-containing solution equalled 3 to 5 per cent of body weight. To control this sodium salts factor, an additional group of animals receiving 3 or 5 per cent of body weight of the identical sodium salts solution, but no colloid, is included in each experiment. Finally, at least one group of animals receiving 15 per cent of body weight of a solution of sodium salts is included in each therapeutic experiment. The solution contained 150 mEq. of Na, one-half as chloride and one-half as acetate.

In viewing these tables, therefore, it is important to compare mortality of controls, mortality with small volumes of sodium salts only, mortality with the various therapeutic

* From the Department of Surgery, New York Medical College, and Flower and Fifth Avenue Hospitals, New York, N. Y. Aided by a Grant from Division of Research and Fellowships, National Institutes of Health, Bethesda, Md.

agents, and finally, the effect of the large quantity (15 per cent of body weight) of sodium salts solution.

Of those animals having a six and a half–second burn (Table I), nearly one-fifth receiving no therapy whatsoever survived. After treatment with whole blood in a volume of 2½ per cent of body weight (equivalent to 1,750 cc. to the average adult) most of the animals survived the first twenty-four hours. Albumin, 1 per cent of body weight (equivalent to 700 cc. of 25 per cent albumin to the average adult), likewise appeared effective and dextran equal to 3 per cent of body weight (equivalent to 2 L. in an adult) appeared nearly as effective. Notice that the large dose of sodium salts was likewise effective.

TABLE I
COMPARATIVE EFFICACY IN BURN SHOCK IN MICE (6½ SECOND SCALD—66°C)*

| Therapy† | No. of Mice | Mortality % | | |
|---|---|---|---|---|
| | | 10 hr. | 24 hr. | 72 hr. |
| None | 16 | 62 | 81 | 81 |
| Blood, 2½% body wt. | 8 | 0 | 13 | 37 |
| Albumin, 1% body wt. | 9 | 11 | 11 | 33 |
| Dextran, 3% body wt. | 10 | 30 | 30 | 50 |
| Sodium, 5% body wt. | 10 | 50 | 60 | 60 |
| Sodium, 15% body wt. | 9 | 11 | 22 | 30 |

* Illustrates comparative results when control mortality is low.
† Therapy given first 5 hr. only.

When the burn was prolonged to eight seconds (Table II), all the untreated animals died within the first twenty-four hours. In this experiment blood and PVP appeared moderately effective, and albumin, globin and dextran less effective. The solution of sodium salts was effective at twenty-four hours but not on subsequent days.

In another experiment using a seven-second burn (Table III) there was again 100 per cent mortality in the control group; and although twice as many animals were present in each of the experimental groups, blood, dextran and PVP did not appear to have any effect whatsoever at twenty-four hours although blood appeared effective at ten hours. In contrast, the 15 per cent of sodium was again effective.

In all these experiments the colloids were injected intravenously in two or three divided doses at one, two and four hours after burn. In many instances some of the animals died before the full course of therapy had been completed. All such animals are excluded. The figures in the table refer only to those animals who received the full course of treatment on schedule. If the animals dying before receiving full treatment were included, the colloids would appear less efficacious.

TABLE II
COMPARATIVE EFFICACY IN BURN SHOCK IN MICE (8 SECOND SCALD—66°C)*

| Therapy† | No. of Mice | Mortality (%) | | |
|---|---|---|---|---|
| | | 24 hr. | 48 hr. | 72 hr. |
| None | 13 | 100 | ... | ... |
| Blood, 2½% body wt. | 5 | 40 | 40 | 40 |
| Albumin, 1% body wt. | 5 | 60 | 80 | 80 |
| Dextran, 3% body wt. | 5 | 80 | 100 | 100 |
| PVP, 3% body wt. | 5 | 40 | 60 | 80 |
| Globin, 3% body wt. | 5 | 60 | 80 | 80 |
| Sodium, 3% body wt. | 10 | 100 | ... | ... |
| Sodium, 15% body wt. | 8 | 37 | 75 | 88 |

* Illustrates some apparent value of blood in one experiment and transient effect of PVP.
† Therapy given first 5 hr. only.

TABLE III
COMPARATIVE EFFICACY IN BURN SHOCK IN MICE (7 SECOND SCALD—66°C)*

| Therapy† | No. of Mice | Mortality (%) | |
|---|---|---|---|
| | | 10 hr. | 24 hr. |
| None | 14 | 100 | 100 |
| Blood, 2½% body wt. | 14 | 57 | 100 |
| PVP, 3% body wt. | 10 | 100 | 100 |
| Dextran, 3% body wt. | 15 | 90 | 90 |
| Sodium, 3% body wt. | 6 | 100 | 100 |
| Sodium, 15% body wt. | 13 | 39 | 53 |

* Illustrates the early transient effect of whole blood and relative lack of efficacy of PVP and dextran.
† Therapy given first 5 hr. only.

As one attempts to summarize the results of these experiments, it would appear that the various colloids tested appreciably reduce mortality during the early hours after extensive burn. This reduction in mortality in the early

hours is significantly greater than can be achieved with a similar small amount of sodium salts only during this early period. It is important, however, to continue the observations beyond the early initial period. It then becomes apparent that none of these colloids significantly reduce the mortality. These results are in agreement with those of other investigators who have made hemodynamic measurements shortly after giving a colloidal infusion in the early hours after injury. When one seeks an answer to the question—Does colloid therapy permit a mouse with a severe burn to survive beyond the initial period?—the answer is *no*. In like manner, the small amount of saline, 3 to 5 per cent of body weight, is insufficient therapy. On the other hand, when the quantity of sodium salts solution was increased to a volume equal to 15 per cent of body weight, a significant number of the burned animals survived the shock period. Similar results have been obtained by Rosenthal and his associates at the National Institutes of Health, with much larger groups of animals in each experiment. They, too, found an early transient beneficial effect from colloids, greater than from similar volumes of saline, but the only therapy that permitted prolonged survival was isotonic sodium salts in a volume equal to 15 per cent of body weight.

While these experiments were in progress, clinical observations were made on severely burned patients using the alternate case method. Despite the cooperation of numerous hospitals and surgeons in this area, the number of severely burned patients available is still too small to permit drawing any conclusions as to the relative efficacy of colloids versus crystalloids in our study. In our small group, however, there have been patients with full-thickness burns ranging from approximately 35 to 55 per cent of body surface based on area requiring grafts, and we would like to show in summary the volume of solution which these patients received. Since these patients survived shock after the extremely extensive burns, the therapy they received may serve to indicate a trend.

In Table IV, which summarizes these results, the age of the patient is shown, as well as the volume of solution administered in treating shock in per cent of body weight.

By the end of twenty-four hours these patients had received solutions of sodium salts, approximately 10 per cent of body weight. The volume of glucose and water or other non-electrolyte solution is excluded from the tabulation. By the end of the period of shock taken as seventy-two hours considerably more electrolyte solution had been administered.

TABLE IV
FLUID THERAPY OF EXTENSIVELY BURNED PATIENTS SURVIVING SHOCK*

| Patient | Age | Weight (kg.) | Colloid† (Given within 24 hr.) | Sodium (Given within 24 hr.) | Colloid (Given within 72 hr.) | Sodium (Given within 72 hr.) |
|---|---|---|---|---|---|---|
| T. | 4 | 15 | None | 8 | None | 29 |
| De. | 5 | 15 | None | 13 | None | 25 |
| O. | 60 | 82 | None | 10 | None | 27 |
| S. | 6 | 17 | 3P | 14 | 3P | 31 |
| K. | 9 | 20 | 4P | 11 | 6P | 31 |
| E. | 30 | 52 | 3P-B | 7 | 7P-B | 13 |
| D. | 38 | 45 | 3B | 7 | 8P-B | 14 |
| G. | 72 | 50 | 1B | 11 | 1B | 19 |

* Full-thickness burn was 35–55 per cent body surface area.

† Colloid and sodium therapy are listed separately. Values for each must be added to obtain total therapy given. Apparently large volumes of Na containing fluids with or without colloid are needed in shock after extensive burns. Volumes are expressed in % body weight. P = plasma; B = blood.

The amounts of colloid given either as plasma or blood or both are also shown; the quantities are small in relation to the total sodium salts solution given. One should note in passing that the sodium content of the plasma used was 190 mEq./L. compared to the normal value of 140 mEq./L. This excess of sodium salts, representing the sodium citrate used in obtaining the plasma, further increased the sodium salts intake and also the intake of bicarbonate precursors since citrate is metabolized to bicarbonate in the body. Thus the ratio of sodium to chloride in the final aggregate of solutions given was approximately the same as that found in normal plasma (1.4:1) and not like that in 0.9 per cent sodium chloride (1:1). These patients came into chemical equilibrium and satisfactory urine outputs were obtained.

The clinical studies indicate that these severely burned patients who survived appeared to require a volume of electrolyte solution similar to that found necessary

for survival in the animal studies. We realize the complexity of this problem and suggest that only by complete and detailed studies of thermal burns in both animals and man can truly effective therapy ultimately be achieved.

## CONCLUSION

At this time our data do not indicate that whole blood or the available colloidal solutions in a volume of 3 to 5 per cent of body weight possess the greatest merit in the treatment of acute, fulminating shock from burns in mice. The significant therapeutic value of solutions of sodium salts, while not yet demonstrated beyond any possible doubt whatever, would seem to reside in the prompt, rapid and systematic administration of a total volume somewhat in excess of 10 per cent of body weight within twelve to twenty-four hours.

*Acknowledgment:* The authors wish to thank Miss Helene Pandelakis and Mrs. Julia Einbinder for their assistance in this work.

## REFERENCE

MILLICAN, R. C., TABOR, H. and ROSENTHAL, S. M. Traumatic shock in mice: comparison of survival rates following therapy. *Am. J. Physiol.*, 170: 179, 1952.

# FAT EMBOLISM*

CAPT. W. W. GLAS, M.C., CAPT. T. D. GREKIN, M.C., AND M. M. MUSSELMAN, M.D.
*Eloise, Michigan*

HUNDREDS of articles have been written on the subject of fat embolism since the 17th century. Still there is great discrepancy of opinion as to its clinical importance, methods for making the diagnosis, its etiology and treatment.[22] How can we resolve the opinion of Warren[27] who believes that fat embolism is an important cause of death and that of Whitson[29] who believes that the diagnosis of fat embolism as a cause of death should be discontinued?

In an effort to clarify some of these points, we have carried out clinical and experimental studies. The results indicate that fat embolism is common in persons suffering from injuries and the effects may be serious. Further efforts should be made to determine the etiology and a specific treatment for fat embolism.

## INCIDENCE AND SIGNIFICANCE

Two fundamental questions which we hoped to clarify were, "How common is fat embolism and how sick does it make people?" We studied 109 patients consecutively admitted to the hospital because of moderate or severe injuries.[15] Twenty-four-hour collections of urine were made daily for fifteen days and examined for free fat. A diagnosis of fat embolism was made in any patient whose urine contained fat. Each patient was examined daily for unexplained pulmonary or cerebral symptoms and x-rays of the chest were taken on all patients whose urine contained fat. Of the 109 patients 52 per cent were considered to have fat embolism, as shown in Table I. Fifty-five per cent of the severely injured and 49 per cent of the moderately injured had fat embolism. Fifty-nine per cent of eighty-two patients with fractures and four of seven patients with burns had fat embolism. Five of twenty patients admitted because of contusions, lacerations or abrasions had fat embolism.

Of the fifty patients considered to have fat embolism, but without injury to the chest, one-third had pulmonary symptoms probably from fat embolism since other cause could not be found. (Table II.)

Of the forty-five persons who had fat embolism but no associated head injury, one-third had cerebral symptoms. One-eighth of the patients had x-ray findings in the lungs compatible with fat embolism. Fat embolism was considered to be a major factor in the death of six of the fifty-seven patients.

TABLE I
INCIDENCE OF FAT EMBOLISM IN 109 INJURED PATIENTS BASED ON PRESENCE OF FAT IN URINE

| Group | No. | Per cent |
|---|---|---|
| All patients | 57/109 | 52 |
| Severly injured | 32/58 | 55 |
| Moderately injured | 25/51 | 49 |
| Fractures | 48/82 | 59 |
| Burns | 4/7 | 57 |
| Contusions, lacerations | 5/20 | 25 |

TABLE II
INCIDENCE OF SIGNIFICANT FINDINGS IN FIFTY-SEVEN PATIENTS WITH FAT EMBOLISM

| | |
|---|---|
| No chest injury (50) pulmonary symptoms | 1/3 |
| No head injury (45) cerebral symptoms | 1/3 |
| Positive chest x-rays | 1/8 |
| Major factor in death | 1/10 |

## DIAGNOSIS

We based the diagnosis in the above study on the demonstration of free fat in the urine. We first compared the test for fat in the sputum with the test for fat in the urine in a group of

* From the Department of Surgery, Wayne County General Hospital, Eloise, Mich. This study was aided by a grant from Chas. Pfizer & Company, Inc.

fifty patients with non-traumatic illnesses.[15] Specimens of sputum and twenty-four-hour collections of urine were made daily for seven days and examined for free fat. Care was taken in the collection and examination of the specimens as recommended by Scott and his colleagues[20] and Scuderi.[23] Effort was made particularly to avoid contamination with oil or fat.

TABLE III
COMPARISON OF TEST FOR FAT IN SPUTUM WITH TEST FOR FAT IN URINE IN FIFTY UNINJURED PATIENTS

| | Positive Sputum | Positive Urine |
|---|---|---|
| 350 Examinations | 111 (32%) | 6 (2%) |
| 50 Patients | 40 | 6 |
| Av. no. days exam. positive | 3 | 1 |

Fat was found in 32 per cent of 350 specimens of sputum, but in only 2 per cent of 350 specimens of urine. Forty of the fifty patients had fat in the sputum on one or more days, but only six of the patients had fat in the urine, and each of these on only one day. (Table III.)

### ETIOLOGY

It is commonly thought that the fat globules gain entrance to the circulation by way of disrupted veins.[27] However, there are those who believe that the fatty emboli arise from a destruction of the emulsion of fat normally present in the plasma. In view of the complex changes which are known to occur with stress,[9,24] we thought it important to determine what effect stress might have on the physiologic emulsion of fat.

In an experiment on rabbits as summarized in Table IV, we simulated the "alarm reaction" by exposure to cold and by the administration of formaldehyde, ACTH* and cortisone.† The animals were sacrificed twenty-four hours after the start of the experiment. We found no remarkable alteration in the turbidity of the serum or in the level of the lipids of the blood. Nor did we find evidence of fat embolism in any of the animals even those primed with 25 cc. of a 40 per cent fat emulsion by gastric gavage.

### TREATMENT

There is no specific treatment for fat embolism. Aside from prophylactic measures, the most important thing is the administration of oxygen. We have investigated the protective value of an emulsifier, Tween 80,‡ against the intravenous administration of 1 cc. of homologous fat in rabbits. The clinically obvious deleterious effects and the pathologic findings were graded from 1 to 3; 1 represented the least abnormality. The results are shown in Table V. While the results were not conclusive, it may

TABLE IV
SUMMARY OF EXPERIMENTAL ALARM REACTION IN RABBITS

| Procedure* | | No. of Animals | Blood Lipids | | Fat Emboli |
|---|---|---|---|---|---|
| | | | Preoperative | Postoperative (%) | |
| Control | | 3 | 387 | +47 | None |
| Cold | 12–30°F. for 24 hr. | 4 | 432 | +25 | None |
| Formaldehyde | 0.4 cc. q. 4 hr. × 4 | 2 | 316 | +28 | None |
| ACTH | 20 mg./kg. q. 4 hr. × 4 | 4† | 428 | +31 | None |
| Cortisone | 20 mg./kg. q. 4 hr. × 4 | 4 | 419 | +15 | None |
| | Total | 17 | | | |

* Blood was drawn for serum lipids at beginning and end of experiment. Animals were sacrificed at end of 24 hours and tissues taken for microscopic examination.

† Two of animals died before end of experimental period. No fat embolism found.

* ACTH was provided by Parke, Davis and Co.

† Cortisone was provided by Merck and Co., Inc.

‡ Tween 80 was provided by the Atlas Powder Company, Wilmington, Del.

be seen that some protection was afforded the animals when Tween 80 was administered intravenously thirty minutes after the fat was given. Even more protection was offered when the Tween 80 was mixed with the fat. Most protection was extended when Tween 80 was administered thirty minutes before the fat was given.

TABLE V
PROTECTION AFFORDED BY EMULSIFIER AGAINST INTRAVENOUS ADMINISTRATION OF 1.0 CC. OF HOMOLOGOUS FAT IN RABBITS*

| Procedure | Degree of Fat Embolism | | |
|---|---|---|---|
| | Clinical | Gross | Micro |
| Control—1.0 cc. fat intravenously | 3.0 (2–4) | 3.4 (2–4) | 2.2 (2–3) |
| 10 cc. 5% Tween 80 30 min. after fat | 2.1 (1–3) | 3.1 (2–4) | 2.0 (2) |
| 10 cc. 5% Tween 80 mixed with fat | 2.0 (1–4) | 2.6 (2–4) | 1.6 (1–3) |
| 10 cc. 5% Tween 80 30 min. before fat | 2.0 (1–4) | 2.3 (2–3) | 1.3 (1–3) |

* Evidence of fat embolism graded from 1 to 3, 1 being the least.

## COMMENTS

*Incidence and Significance.* We know of no other study in which an effort has been made to screen a group of injured patients for the presence of fat embolism. Since many articles on fat embolism consist of reports of an individual case or a few cases, most of whom died, it must be that most physicians consider it to be a rare but fatal entity. Harris and his coworkers[6] report six cases, three of whom died. Wilson and Salisbury[30] found clinical evidence of fat embolism in only 8 of 1,000 consecutive battle casualties including 119 with fractures of long bones, but six of the eight died. Five of seven patients described by Newman[16] died.

Vance[25] found at autopsy that 75 per cent of persons with fractures of long bones had fat embolism. In 20 per cent he considered it to have been a significant factor in causing death. Robb-Smith[19] concludes from his study that fat embolism is an almost constant finding in deaths following accidents and in many cases was probably a major factor in the fatal outcome.

Wyatt and Khoo[31] demonstrated fat emboli in each of thirty subjects who died after injury. None was found in fifteen cases of nontraumatic sudden death. Of twenty-two dying after a lingering illness only one, who died three weeks after extensive burns, had embolic fat. Denman and Gragg[2] found nine cases of fat embolism at autopsy in nineteen persons dying after injury. In six of the nine it was sufficiently severe as to be considered the cause of death. Mallory[12] found fat embolism at autopsy in forty of sixty battle casualties of the last war; in ten it was extensive.

In a recent study of soldiers killed in Korea[4] fat embolism was found in 72 per cent and it was of a significant degree in 33 per cent. It was thought to be an important factor in causing death in 13 per cent of the entire group of seventy-six autopsies or in 40 per cent of those having a significant degree of fat embolism.

Lehman[11] believes that the finding of free fat in the sputum and urine may be clinical evidence of fat embolism, but it is essential to differentiate between fat embolism, the pathologic entity, and fat embolism, the clinical state. Although the former is common, as shown by clinical testing and postmortem examination, the latter is rare.

On the basis of our findings, we agree with Elting and Martin[3] and Scuderi[23] that fat embolism is a condition of great importance and of relatively frequent occurrence. Most cases reported have been fatal. There are probably many more with mild symptoms which recover and are never recognized. This explains the difference of opinion as to the incidence of and the mortality from fat embolism.

Among casualties of the fighting in Korea[4] severity of injury showed little relation to the presence or severity of fat embolism. This agrees with the results of our study.

Although fat embolism has been found most frequently after fractures, it has been reported as occurring after burns and other soft tissue injuries by Wakeley,[26] Warren[27] and others.[4,22]

*Diagnosis.* Fat embolism should be suspected in every patient who has been injured and exhibits characteristic clinical manifestations, no other cause for which can be demonstrated.

Various tests have been devised to verify the diagnosis of fat embolism. Scriba[21] described the demonstration of fat globules in the urine as a method of establishing the diagnosis in 1880. Scuderi[23] has described the care which should be taken in the collection and staining of specimens. Elting and Martin[3] thought it probable that a careful examination would always reveal fat in severe cases and it might be present in mild cases. The value of the test has been questioned, but the worst criticism has been that in cases of fat embolism fat will be found in the urine late or not at all.[16,19,27] We found fat in the urine as early as the first day after injury. We are convinced that when properly carried out the test for fat in the urine is a practical and reasonably accurate test for fat embolism.

Warthin[28] described the demonstration of free fat in the sputum as a test for fat embolism in 1913. The technic is described by Scott and his co-workers[20] who warn that fat should not be expected in the sputum until thirty-six hours after the onset of pulmonary symptoms. We agree with Scuderi[23] and Nuessle[17] that the test is positive so frequently in the absence of fat embolism as to make it unreliable.

Scuderi[23] describes the change in roentgenograms of the chest in experimental fat embolism and suggests their value in clinical cases. Scott and his associates[20] agree that they may be of assistance, but state that a considerable quantity of fat may be retained in the lungs causing respiratory embarrassment without producing radiographic changes.

Newman[16] mentions lung puncture as a possible diagnostic aid which has been carried out in some cases. This would seem to be a hazardous procedure. Kershman and Haddad[10] discuss the changes found in cases of fat embolism by electroencephalographic examination by themselves and others.

Scuderi[23] discusses the quantitative determination of fat in the blood and of the surface tension of the blood which he considers to be of no value. He speculates that the dark field examination of the blood for microscopic droplets of fat should be of great aid but this is not proved.

*Etiology.* Lehman[11] contends that the traditional mechanism of production of fat embolism cannot be accepted because (1) fat is not freed by trauma in sufficient quantity to produce symptoms and (2) there is no logical mechanism for the entrance of fat into the blood. He offers theoretical and experimental evidence to support the theory that embolization is caused by destruction of the physiologic emulsion of fat in the circulating plasma by physical and chemical changes in the blood.

Warren[27] believes that there is no convincing evidence that chilomicrons coalesce to form masses able to produce fat embolism. If it is caused by non-specific stress as suggested by Lehman,[11] it should be common after operations. Yet it is not so reported and we have not found it so in exploratory studies. Montanus and his colleagues[14] found no typical pattern of change in lipoid levels of the serum in patients subjected to various surgical procedures under a variety of anesthetic agents including ether. Under the conditions of our experiment simulating stress, there is no evidence that either a change in the emulsion of circulating fat or fat embolism is a part of the pituitary-adrenal reaction to stress.

Particles of marrow can gain entrance to the circulation as discussed by Rappaport and his colleagues[18] and it would seem plausible that globules of fat would enter even more easily.

The discrepancy between the amount of fat released by trauma and that necessary to cause symptoms as determined experimentally has been explained in several ways. Harris and his associates[6] concluded from experimental work that the serious pathologic changes produced by fat embolism are probably due in part to the freeing of fatty acids by the action of tissue lipase. Harman and Ragaz[5] also believe that a chemical change in the fat with the release of irritant substances is probably an important factor. Scuderi[23] considers oleic acid, one of the split products of neutral olein of bone marrow, to be the probable lethal factor. However, both Warren[27] and Denman and Gragg[2] demonstrated by staining technics that the fat in emboli of persons dying from injuries was present as neutral fat and not hydrolyzed. There was nothing in the appearance of the tissues to indicate any appreciable breakdown of fat or that fat was in any way toxic to either vascular endothelium or formed elements of the blood.

Hess[8] concludes from his experimental work that reduced activity of the lipase of the plasma and lungs is an important factor in the development of fat embolism.

*Treatment.* Theoretically, an emulsifier

should be effective in the treatment of fat embolism because an emulsifying agent should reduce the oil-water interface. As discussed by Davis and Goodchild[1] it is the relatively massive oil-water interfaces which make the emboli behave like solid obstructions in the vessels.

There are hundreds of emulsifying agents. Scuderi[23] used gardinol®* (sodium lauryl alcohol sulphonate) in the treatment of experimental fat embolism in dogs and concluded it was of no value as a therapeutic agent. Although his experiments, as ours, were not conclusive, they do suggest that further trial of other emulsifiers might uncover one which is effective in the treatment of fat embolism.

Herrmann[7] found by experimental studies in animals that the intravenous administration of a dextrose-alcohol mixture was effective in relieving the symptoms and reducing the fat in the capillaries of the lungs. Monson and Dennis[13] used choline chloride experimentally in dogs and found it effective. They report striking results in clinical cases of fat embolism.

Scuderi[22,23] and Robb-Smith[19] discuss the use of sodium carbonate, decholin® sodium and various other drugs and technics, all of which have been found to be either dangerous or ineffective in the treatment of fat embolism.

## SUMMARY

On the basis of the presence of fat in the urine, we found fat embolism in half of 109 persons moderately or severaly injured. The incidence after moderate injuries did not differ significantly from that after severe injuries. It was most common after fractures and burns but occurred in about one-fourth of other soft tissue injuries.

The demonstration of free fat in the sputum was found to be an unreliable test for fat embolism.

Neither a change in the emulsion of the circulating fat nor fat embolism occurred after a simulated "alarm reaction" in rabbits.

Tween 80 did not have a significant effect in the treatment of fat embolism in rabbits.

## CONCLUSIONS

Fat embolism is a condition of great importance and of relatively frequent occurrence. It should be suspected in every patient who has been injured and exhibits characteristic clinical manifestations, no other cause for which can be demonstrated.

The test for fat in the urine is a practical and reasonable accurate test for fat embolism.

The issues concerned in the pathogenesis of fat embolism are far from settled.

Further search should be made for an effective specific treatment for fat embolism.

* A product of E. I. du Pont de Nemours & Co.

## REFERENCES

1. Davis, H. L. and Goodchild, C. G. Emulsion stability and fat embolism. *J. Chem. Education*, 13: 478, 1936.
2. Denman, F. R. and Gragg, L. Fat embolism; a diagnostic enigma. *Arch. Surg.*, 57: 325, 1948.
3. Elting, A. W. and Martin, C. E. Fat embolism with study of two fatal cases. *Ann. Surg.*, 82: 336, 1925.
4. Fat embolization. Annual Publication, Historical Report, 406th Medical General Laboratory, Professional Section, Tokyo, 1951, p. 92.
5. Harman, J. W. and Ragaz, F. J. Pathogenesis of experimental fat embolism. *Am. J. Path.*, 26: 551, 1950.
6. Harris, R. J., Perrett, T. S. and MacLachlin, A. Fat embolism. *Ann. Surg.*, 110: 1095, 1939.
7. Herrmann, L. G. Effect of dextrose-alcohol mixture upon pulmonary fat embolism. *Proc. Soc. Exper. Biol. & Med.*, 30: 558, 1933.
8. Hess, W. Fat embolism and lipases. *Helvet. chir. acta*, 15: 163, 1948.
9. Ingle, D. J. Biologic properties of cortisone. *J. Clin. Endocrinol.*, 10: 1312, 1950.
10. Kershman, J. and Haddad, F. Cerebral fat embolism. *Canad. M. A. J.*, 66: 95, 1952.
11. Lehman, E. P. Fat embolism. *Internat. Surg. Digest*, 7: 131, 1929.
12. Mallory, T. Cited by Dunphy, J. E. and Ilfeld, F. W. Fat embolism. *Am. J. Surg.*, 77: 737, 1949.
13. Monson, E. M. and Dennis, C. Therapeutic effect of choline chloride in dogs with fat emboli produced by bone marrow curettage. *Proc. Soc. Exper. Biol. & Med.*, 70: 330, 1949.
14. Montanus, W. P., Ames, A. and Herrmann, L. G. A study of the lipemia curve following surgical operations under ether anesthesia. *Surgery*, 2: 401, 1937.
15. Musselman, M. M., Glas, W. W. and Grekin, T. D. Fat embolism, a clinical investigation. *Arch. Surg.*, 65: 551, 1952.
16. Newman, P. H. Clinical diagnosis of fat embolism. *J. Bone & Joint Surg.*, 30B: 290, 1948.
17. Nuessle, W. F. Significance of fat in sputum. *Am. J. Clin. Path.*, 21: 430, 1951.
18. Rappaport, H., Raum, M. and Horrell, J. B. Bone marrow embolism. *Am. J. Path.*, 27: 407, 1951.
19. Robb-Smith, A. H. T. Pulmonary fat embolism. *Lancet*, 1: 135, 1941.
20. Scott, J. C., Kemp, F. H. and Robb-Smith, A. H. T. Pulmonary fat embolism; clinical and radiological observations. *Lancet*, 1: 228, 1942.
21. Scriba, J. Untersuchungen über die Fettembolie. *Deutsche Ztschr. f. Chir.*, 12: 118, 1880.

22. Scuderi, C. S. Present status of fat embolism. *Internat. Surg. Digest*, 18: 195, 1934.
23. Scuderi, C. S. Fat embolism; a clinical and experimental study. *Surg., Gynec. & Obst.*, 72: 732, 1941.
24. Selye, H. General adaptation syndrome and the diseases of adaptation. *J. Clin. Endocrinol.*, 6: 117, 1946.
25. Vance, B. M. Significance of fat embolism. *Arch. Surg.*, 23: 426, 1931.
26. Wakely, C. P. G. Treatment of war burns. *Surgery*, 10: 207, 1941.
27. Warren, S. Fat embolism. *Am. J. Path.*, 22: 69, 1946.
28. Warthin, A. S. Traumatic lipaemia and fatty embolism. *Internat. Clin.*, 4: 171, 1913.
29. Whitson, R. O. A critique of fat embolism. *J. Bone & Joint Surg.*, 33A: 447, 1951.
30. Wilson, J. V. and Salisbury, C. V. Fat embolism in war surgery. *Brit. J. Surg.*, 31: 384, 1944.
31. Wyatt, J. P. and Khoo, P. Fat embolism in trauma. *Am. J. Clin. Path.*, 20: 637, 1950.

## DISCUSSION OF PAPERS BY DRS. FOX, MERSHEIMER, LASKER AND WINFIELD, AND BY GLAS, GREKIN AND MUSSELMAN

Grover C. Penberthy (Detroit, Mich.): I will limit my remarks to Dr. Musselman's paper. It is interesting to note that in his study it is claimed that fat embolism is more common than many of us are inclined to believe. His experience is taken from a comparatively large service in the Wayne County General Hospital, which is midway between Detroit and Ann Arbor. There are many highways in the neighborhood and many accident cases are taken to Wayne County General Hospital.

I recall that several years ago we had a few cases at Harper Hospital in Detroit, and the complication was given considerable study, but of late we think little of fat embolism. Dr. Musselman has shown that it is more common than we are inclined to believe, and the condition or complication may be serious.

I would also call attention to the receiving hospital service in Detroit. Dr. Cameron is here, and I wonder if he would be kind enough to contribute to this discussion.

I would like to ask Dr. Musselman whether or not diagnoses of post-traumatic complications such as post-traumatic psychoses and delirium tremens could not, by chance, be fat embolism but not recognized as such. I think it is important that such studies be brought to our attention because, while they may not amount to very much, they are nevertheless very serious in their complications.

Duncan A. Cameron (Detroit, Mich.): I think the presentations which Dr. Musselman has given us in the last couple of years on these studies of fat embolism have been interesting to all of us. I am sure on our service at Receiving Hospital, where we have not gone into this problem of fat embolism, that in the course of every year we must lose patients for which we have no obvious explanation, simply because of this entity. I would be interested in having Dr. Musselman tell us as he closes just how much a problem it is to discern in these injured people the presence of an excess of fat in the urine from the urinary studies. If it is a matter which can be easily accomplished, I think from the evidence he has presented all of us should carry out those studies, and we will be in less of a quandary to explain death in our traumatic patients.

Kellogg Speed (Highland Park, Ill.): I wonder why we should pin our faith on one symptom in diagnosing fat embolism, and why we should depend so much on the lipuria found. It seems to me one would have to compare the urine of 10,000 ordinary admissions or postoperative patients with that of 10,000 traumatic patients in order to arrive at some comparative basis on the lipuria side alone. Secondly, I would like to hear something said about the pathologic condition in the fatal cases. What was the pathologic status in the brain, the lungs, heart, heart valves, and so on?

I think back to McCountsworth in Chicago, where many years ago at the County Hospital he made a most meticulous and careful study with histologic preparations showing the capillaries in various parts of the body packed or blocked with fat droplets, and so on, putting the thing on a really pathologic basis. I would like to hear something said about that by the essayists.

William H. Cassebaum (New York, N. Y.): My interest in fat embolism was aroused by a talk by Dr. Harris of this Society in 1939. In the course of a year we found six cases. That does not mean the fatalities in these cases were due to fat embolism, but the patients had moderate to severe degrees of fat embolism. The pathologic material was obtained from Dr. Vance, of the Medical Examiner's office of this city, who wrote a paper on this condition and studied a large number of cases (100 or more). He found there was an incidence of fat embolism I believe somewhat over 50 per cent, but that the number of fatalities that could be attributed to fat embolism was relatively low.

I think the literature also cites as a diagnostic aid in the determination of fat embolism the presence of petechiae in the skin, particularly in the eyegrounds. Also, a suggestive symptom is a fall in the blood count in severe cases. Of course, that symptom may now be obscured by the multiple transfusions which we give traumatic cases. Fatal cases have been attributed to pulmonary involvement, which simulates bronchial pneumonia or atelectasis, and the cerebral involvement which simulates delirium tremens or head injury. It was Dr. Vance's opinion that none of the fatal cases was due to fat embolism in the kidneys. While fat

embolism does occur in the kidneys, they were not involved to a degree which would cause fatality from anuria.

JAMES MITHOEFER (Cooperstown, N. Y.): I want to thank Dr. Fox for his very interesting observations on burned mice. I wonder if his burned mice that received the excess sodium had free access to drinking water during the period of the experiment and also, if they had, whether the amount of water they drank was measured. If the water intake of these mice was in fact increased, it would make the mechanism of these results somewhat more clear in my mind, because then those mice would have available materials to fill up the tremendously expanded extracellular space which we know occurs in the twenty-four hours after burns.

M. M. MUSSELMAN (closing): First, I will answer Dr. Speed. The question of the presence or absence of fat in the urine was used in these cases as a screening procedure, and it is not in any sense to be construed as the only diagnostic measure to be applied in patients suspected of having fat embolism. We observed these patients for other signs and symptoms.

Of the six fatal cases that we thought were due to fat embolism, three passed to the coroner in Wayne County, and we have no idea what he found as far as fat embolism is concerned. The three cases on which we had the privilege of carrying out an autopsy showed fat embolism in the capillaries of the lungs, the brain and the kidneys. It was interpreted by our pathologist as being of significant degree. By "significant" I mean possible of being a major factor in the cause of death. Also, we considered these deaths due to fat embolism because other good reasons for the fatal termination could not be found.

Answering Dr. Penberthy, and it has already been commented on by the other discussers, fat embolism is frequently confused with delirium tremens, post-traumatic psychosis, pulmonary contusion, cardiac contusion, bronchopneumonia and surgical shock. These latter diagnoses often get the credit for the morbidity and mortality which the patient suffers.

In regard to Dr. Cassebaum's remarks, I can only say that we watched carefully for petechiae in our patients. Because we found a relatively larger number of patients whom we considered to have fat embolism, the incidence of petechiae was less than generally described. Petechiae were observed in only two of our patients.

In regard to the anemia which has been described by Harris, Perrett, and McLachlin as another diagnostic criterion for fat embolism, we observed no anemia in our patients which could not be ascribed to other causes. We were not able to substantiate their opinion that anemia is an important criterion for this syndrome.

CHARLES L. FOX (closing): The answer to Dr. Mithoefer's question is: No. The mice did not receive water for the first fifteen to twenty-four hours following the infusion of 15 per cent of body weight of sodium salt solutions. As a matter of fact, that is a rather important point because in studies of patients with burns, one of the persistent observations is that the plasma sodium becomes fairly low. Apparently it is one of the confusing things in the minds of many people that water or dextrose in water serves to make up extracellular fluid. The solution given, 0.15 molar sodium, is isotonic. It is the normal composition of extracellular fluid and does not need water to form extracellular fluid. It is itself the composition of extracellular fluid. In the plasma-treated patients, many of whom did receive dextrose in water, it is rather interesting that the plasma sodium in the plasma used was extremely high, running about 190 mEq./L., with normal plasma value, 140. This extra sodium came from the sodium citrate added to the plasma when prepared; and in the calculations apparently a lot of this sodium made up for some dextrose in water. In circumstances in which hypertonic sodium is given in one way or another, no additional water is given.

# TETANUS REPORT OF TWO FATAL CASES FOLLOWING TRAUMA

WALTER SCOTT NETTROUR, M.D.
*Pittsburgh, Pennsylvania*

THE manifestations of tetanus are so dramatic that it has been recognized as a clinical entity for centuries. Both the regional and systemic forms of the disease are clearly described in the writings of Hippocrates.

Tetanus occurs in virtually all warm blooded animals and is found where soil is contaminated by human or animal excreta. Recent inquiry[1] into the occurrence of tetanus spores on the pavement of Baltimore shows an incidence of 17 per cent.

Tetanus may occur in minor wounds and has followed the contamination of the umbilicus in the newborn, to the prick of a needle in the grandmother. Spores may be carried by dust storms and have become a problem in certain sections.

Injuries in which tetanus contaminations are common are: (1) compound fractures, (2) wounds with blank cartridges and (3) imbedded splinters and puncture wounds of the feet. Bee stings, insect bites, burns, hypodermic injections and bed sores are frequently forerunners of tetanus. Two cases[2,3] have been reported following minor nose injury and Pratt[4] stated no demonstrable portal of entry could be found in 20 to 30 per cent of the cases.

*Bacteriology.* Clostridium tetani is a slender, motile rod, two to five microns in length, which tends to grow singly and with spherical spores which give a characteristic squash-racquet appearance.

The mere presence of spores is not sufficient to cause tetanus. There must be some tissue injury or infection to produce clinical manifestation. There must be tissue necrosis to facilitate the growth. The spores germinate only when there is decreased oxygen tension. The spores of tetanus are very resistant and killed at 131°C. Boiling will not kill and it is necessary to use 15 pound pressure in the autoclave for twenty minutes.

The toxin of Clostridium tetani is probably the most potent water soluble poison known to man. One one-millionth of a cc. will kill a 20 gm. mouse in two hours. Vigorous treatment is necessary because one four-hundredth of the usual intravenous lethal dose given intrathecally can cause death; and once a lethal dose of tetanus toxoid is fixed in the central nervous system, no amount of tetanus antitoxin will help.

Tetanus multiplies in a contaminated and not in a clean wound. The presence of Clostridium tetani in a sterile wound will not cause tetanus. Water soluble toxin often causes cramps and contractions in adjacent local muscles for months and then returns to normal.

First, Clostridia tetani multiply in an injured or contaminated wound and then the toxin passes into the blood and lymph to the central nervous system. Specific cells in the medulla and motor end organs react to toxins. It forms a secondary substance not neutralized by antitetanic serum. The secondary substance circulates in the blood and seems to interfere with some essential part of the respiratory mechanism. Once a lethal amount of this hypothetical substance has been formed, no known treatment will save the life of the animal.

The incubation period of tetanus is from five to ten days. It may be as short as three days or as long as three weeks. Tetanus occurs in two forms in man, a generalized and a localized form. In the latter, a group of muscles show local unyielding rigidity which persists for weeks or months and leaves no residual change. Occasional general tetanus develops despite antitetanic serum. This usually occurs in two to three weeks.

Onset is usually ushered in by stiffness of the jaw, with restlessness, irritability and dysphagia. Stiffness of the extremities, headache or fever may occur. Later, trismus (masseter rigidity) with a sardonic smile (characteristic appearance from spasm of the facial muscles and involuntary raising of the eyebrows), and contraction of the back muscles may become so great that opisthotonos occurs. Rigidity of the

abdominal muscles has led to errors in diagnosis. Dysphagia is often severe and interferes with nutrition. Cyanosis and asphyxia from spasm of the glottis and respiratory muscles may occur. Death occurs from spasm of the respiratory muscles but sometimes from no apparent cause.

The death rate is 50 to 70 per cent. In some no treatment will aid; in others, no treatment is necessary. There is an intermediate group in which prompt diagnosis and adequate treatment determines the issue of life and death. A long incubation period usually permits a better prognosis. When antitetanic serum is given the disease is usually mild and the chance of recovery is good.

*Treatment.* Intrathecal, intramuscular and intravenous tetanus antitoxin is administered, as well as tetanus antitoxin injected at the site of the local lesion. Rest, oxygen, fluid and electrolyte balances are maintained. Active immunization by toxoid has the benefit of not only protecting in minor injuries or in places where medical aid is not available, but also of decreasing morbidity and occasional mortality from serum sensitivity.

*Immunology.* Tetanus is a preventable disease. W. M. Firor[1] stresses that the importance of immunizing children, farmers, artisans and military personnel cannot be overemphasized. The military services have shown the value of routine compulsory immunization. In five million personnel of the U. S. Navy during World War II[5] only four cases of tetanus occurred, two of which terminated in death and one of these deaths was due to an infection developing as a result of an ingrown toenail which had been incurred before entry into military service. Of the two fatal cases, one had no record of immunization and the other had an immunization record which was incomplete. That danger exists for the unimmunized is apparent from the fact that during these same years the number of deaths due to tetanus, reported to the National Office of Vital Statistics as having occurred within the United States among civilians, was 2,574. At this rate it would be expected that there would be about twenty-five deaths among the five million officers and men who served in the U. S. Navy and Marine Corp during World War II. Ample evidence of the effectiveness of the prophylactic program in the Navy is seen in the incidence rate of 0.40 per million in World War II to 4.77 in World War I and 3.34 in intervening years (1920 through 1941).

The Army also started active immunization in 1941 and during World War II had twelve cases of tetanus,[6] only two of which had received a full immunization course of injections. This compared favorably to the seventy cases of World War I. At the same time among civilians at Manilla, there were at least 473 cases with 389 deaths.

During the Normandy invasion over eighty cases of tetanus were reported in the unimmunized German ground forces, but no cases in the immunized Luftwaffe.

There can be no doubt of the efficacy of this procedure. Full value of active immunization necessitates three injections of 1 cc. of tetanus toxoid in three-week intervals and 1 cc. reinforcing stimulating injection a year later. An emergency stimulating dose of 1 cc. centimeter is given at the time of injury. American industry should duplicate the fine record of our military service in saving lives. They should adopt a like or similar immunization program as used in our armed forces to prevent tetanus. This is known to immunize for three years and it is believed that longer immunization is obtained with a booster injection given at the time of any injury. Such an active immunization program is being organized in one of Pittsburgh's industries.

## CASE REPORTS

CASE 1. A fifty year old male carpenter was admitted to the Western Pennsylvania Hospital, Pittsburgh, Pa., on April 3, 1947, following a traumatic amputation of the distal centimeter of the right index finger tip. The finger had been injured by a saw just prior to admission while the patient was doing repair work inside a library. The wound was open and had bled profusely. Because of the free bleeding from the open abrasion and the history of an indoor injury, no tetanus antitoxin was given. A thin, partial thickness graft was applied under local anesthesia. Convalescence was uneventful and the patient was discharged with a clean healed wound nine days after admission.

Fifteen days after the original injury, April 18th, the patient's family called, saying he was having some difficulty in swallowing. He was rushed to the hospital by ambulance. Intramuscular and intrathecal tetanus antitoxin (30,000 units each) were administered on admission and this was repeated every sixth hour for a total of 500,000 units. The patient was highly excited and suffered muscle

clonus, respiratory difficulty and pulmonary edema. He was placed in oxygen and his excitation and spasm were controlled by intravenous barbiturates. He was also maintained on food and fluid by the parenteral route. There was no cyanosis, and suction controlled the intratracheal secretions and the anesthesia consultant believed a tracheotomy was not indicated. For two days he showed moderate improvement and then suffered a rapid decline and death.

CASE II. A fifty-four year old male was admitted on April 20, 1952, to St. John's Hospital, Pittsburgh, Pa., as an emergency following a gasoline flash explosion. The accident occurred while using gasoline in his yard for the eradication of ants.

He was treated with sterile boric pressure dressings and received 1,500 units of antitetanic serum for prophylaxis. There were second and third degree burns of both upper extremities, anterior chest and abdomen.

The patient appeared to be doing fairly well with the exception of developing a flexion contracture of the left wrist due to the depth of the burn. On May 10, 1952, twenty days after his accident, he was given a light manipulation of this extremity under pentothal anesthesia and the left wrist and forearm were splinted in a neutral position.

Three days after this minor procedure the patient complained of difficulty in swallowing. He was given 300,000 units of tetanus antitoxin but he had a rapid decline. The patient showed no skeletal muscle spasm but progressive severe dysphagia and a general overwhelming toxicity. There was no respiratory embarrassment or undue excitation. The patient had been given penicillin therapy since admission on the day of injury. In spite of intensive treatment he expired thirty-six hours after his first symptoms. The four physicians who saw the patient in consultation believed he expired from clinical tetanus.

## SUMMARY

1. Tetanus is a preventable disease.

2. Two fatal cases of tetanus have been reported following trauma, one of which had received a prophylactic injection of antitetanic serum.

3. A review of the results of prophylaxis in the Armed Forces during World War II is compared with the civilian population, and also with the incidence in unimmunized enemy troops and civilians in combat zones.

4. Industry should organize active routine immunization to prevent tetanus.

## REFERENCES

1. FIROR, W. M. Cecil and Loeb's Textbook of Medicine, 7th ed., p. 195. Philadelphia, 1951. W. B. Saunders.
2. SPAETH, R. A. Clinical study of tetanus. *Am. J. Dis. Child.*, 60: 130, 1940.
3. TUDOR, R. B. Tetanus resulting from foreign body in the nose. *J. A. M. A.*, 149: 660, 1952.
4. PRATT, E. L. Clinical tetanus: a study of fifty-six cases with special reference to methods of prevention and a plan of evaluating treatment. *J. A. M. A.*, 129: 1243, 1945.
5. Statistics of Navy Medicine, vol. 3, no. 12, p. 12, December, 1947.
6. LONG, A. P. and SARTWELL, P. E. Tetanus in the United States Army in World War II. *Bull. U. S. Army Med. Dept.*, 7: 371–385, 1947.

## DISCUSSION

RALPH G. CAROTHERS (Cincinnati, Ohio): It appears to me that we must not forget that tetanus will be manifested as a local reaction for a while in a great many cases. I sometimes like to think that it is in all cases. If one goes back into any case that comes in with lockjaw, he can get a history of muscle cramps or spasms in the limb involved, perhaps over a period of some days before the jaws themselves became locked. This is a very important point because, being overlooked by surgeons and house staff, it sometimes has permitted them to miss the boat entirely and to allow the patient to become a casualty when he might have been saved. It was horrible in our own hopsital when one of the surgeons, handling a case with a compound fracture, observed these muscle contractures. He called in a psychosomatic specialist. This boy died because this specialist insisted that it was hysteria, that he had some kind of nerve block from the way his mother changed his diaper or something like that. They let the thing go to a full-blown tetanus. They should have seen this local tetanus and caught it immediately.

One other point: In our part of the country we still have the old-fashioned Viennese beer gardens and it is the common practice to have the grounds strewn with tanbark. Tanbark is a very common source of the spore-bearing, oxygen-free bacteria. Remember that in any of those wounds in which tetanus may be present, the gas gangrene spores may also be present. I have had two cases in which both diseases ran at the same time and we cured both of them. Therefore, in prophylaxis if you are giving the horse serum at all, I think the gas gangrene serum should be given as well as the antitetanic serum.

THOMAS BARTLETT QUIGLEY (Boston, Mass.): Dr. Nettrour's paper was of particular interest to me because one of my pleasanter duties is the care of injuries and surgical ailments of a large group of young men in an academic environment. These young men generally fall into three groups: One group has been in the Armed Services, another group we know has had good pediatric care, and one can rest easily about those two because they

have been actively immunized. It is the third group that worries me and my colleagues. We do not know and the patients do not know whether or not they have ever had active immunization.

As Dr. Nettrour pointed out, even tiny and insignificant wounds occurring at the most unlikely places can produce tetanus. I have seen tetanus resulting from a tiny cut on the sole of the foot from a broken clam shell on a sun-drenched beach.

What are we to do with this third group who present minor breaks in the skin? We are not at all happy about passive immunization because the incidence of allergic reaction is high. It seems that in our small infirmary there are always one or two men with hives or large joints, and our medical confreres are worried about the future of these young men with regard to renal function and such other complications.

There is also some evidence, I have been informed, that 1,500 units of tetanus antitoxin is inadequate, and that would certainly seem to have been brought out by Dr. Nettrour's paper. I would like to ask him what we should do with this third group.

An experiment which I carried out in this connection may be of interest. For many decades the playing fields of this university were taken care of by horse-drawn mowing machines, and only in relatively recent years have these been supplanted by tractors. Immediately after the war, when tetanus was fresh in my mind from having seen it in German prisoners-of-war, I collected some fifteen or twenty samples of earth one afternoon from the various sections of the playing fields. These were turned over to a good friend who was in the department of bacteriology at Harvard and I asked him to find some spore-bearing organisms. He worked for several months and found not one single spore-bearing organism. This is of interest in connection with a note I saw in Time Magazine to the effect that the playing fields of Eton are now so heavily infested with tetanus spores that it is a serious matter to those concerned with the health of Etonians.

Harold G. Kuehner (Pittsburgh, Pa.): In any case report it is always difficult to do more than draw a certain inference of agreement or difference with the essayist. With regard to Dr. Nettrour's first reported case, it seems that if there is not a positive history of previous immunization with toxoid, the use of an adequate prophylactic dose of tetanus antitoxin is advisable. Secondly, in this particular case it would appear to me that there would have been a place during the therapeutic management of this patient for tracheotomy; whether or not it would have made any difference in the ultimate outcome, it certainly would have made a difference in the comfort of the patient.

With regard to the second case, there is at least in my mind a definite confusion as to the differential diagnostic positiveness in the individual. In other words, this was a late onset. I believe Dr. Nettrour reported it was twenty days after receipt of his injury. He did have a 1,500 unit prophylactic dose of tetanus antitoxin. He had at the same time, upon his admission to the hospital, the administration of fairly large doses of penicillin, I believe 400,000 units twice a day routinely. With the onset of his dyscrasia and his intoxication he was given massive doses of tetanus antitoxin. Within thirty-six hours after the onset of his difficulty in swallowing, the patient had expired.

Inasmuch as he was seen by four consultants, the diagnosis of tetanus is probably the correct one. However, there are certain potential confusing factors entering into the differential diagnosis which must be thought of. To repeat what Dr. Quigley has stated, I think there is quite general agreement that 1,500 units are not a sufficiently large dose for prophylactic therapy. I do not know that there is any uniform agreement, but we have made it a practice to use 10,000 units on our own service.

There is another detail that I have gleaned from this second case report, namely, is it possible that with the introduction of the antibiotics, in this case as in many other similar local and systemic infections, we have produced a masking of symptoms, with a tendency to obscure or to make the diagnosis more difficult?

Finally, inasmuch as there is such a positive effect from toxoid immunization and inasmuch as our pediatricians are doing an excellent job in the younger group, it might be intriguing for many of us, who have the responsibility for the health welfare of large organizations, to consider encouraging the use of mass immunization in your employees.

Laurie H. McKim (Quebec, Canada): I happen to be one of the older generation who has seen a good deal of tetanus. There are two points particularly to which I want to refer:

One, which has not been mentioned by anybody, is the incidence of tetanus in diabetic gangrene. I have had one case personally. The case was spotted by the general practitioner who saw it first as a localized lesion. He sent the case in to the hospital with the diagnosis of tetanus. Only two of the toes were involved. There was very definite spasm of the leg. I agree with Dr. Carothers and with those men who have seen a good deal of this condition that tetanus is always at first a localized lesion.

The second point that I wish to bring up is the importance of intrathecal administration. In the first war we saw a great deal of tetanus. I remember that once in our general hospital we had three patients with very severe tetanus, all of whom eventually recovered. All of them were under treatment for a period of nearly three weeks, getting antitetanic serum by all methods that could be devised.

In all those cases there were times when we thought we were going to lose the patients and when we had to give them chloroform anesthesia in order to do a spinal puncture to give them the intrathecal administration of tetanus antitoxin. The importance of that is perhaps forgotten at the present time. In many patients who you think are dying and are in opisthotonos, you will find, if you give them a small amount of anesthesia and an intrathecal administration, they come out of it beautifully. I am sure that we cannot overemphasize the importance of administration by this method.

THORBURN S. MCGOWAN (Boston, Mass.): In the Marine hospitals of the Public Health Service tetanus occurs with not too much regularity, but enough for us to worry about. As you know, we take care of a number of seamen of the merchant group, most of whom are from this country, some of whom are foreign seamen. In the last few years it happens that both at Seattle, Washington, and at Boston I have supervised the handling of three cases of tetanus, and there are one or two lessons that have not been emphasized up to the present point which I think should be emphasized.

One has already been mentioned by Dr. Kuehner, and I hope this brings out the advisability of early tracheotomy. There are a couple more which I think certainly are basic and which we should consider, one of which is eradicating the focus. It is my understanding that as long as a focus of tetanus persists in the body in a place usually where our antibiotics will not do too much good in getting to it because of the barrier of granulation tissue or abscess wall, it will continue to produce the toxin. It is also my understanding that this is a neurotoxin and that it is the effect of toxin which kills. Therefore, we should try to eliminate the focus.

The first of these three cases was a man who had a right colon resection. About nine days later he began to show symptoms which were interpreted as tetanus. We did not do what should have been done, re-explore him, because his bowel continuity had been restored. He died in a laryngeal spasm at the end of about eight days of treatment, during which time we thought he was recovering because he had gone beyond the extremely excitable stage and had gotten to the point at which we were controlling him rather well. He died suddenly, and without a tracheotomy. At autopsy he had a very small abscess from which a pure culture of tetanus bacillus was grown, and this obviously proved the case. The suture material in that case was catgut.

The next case was a man who had a knife wound sutured in Alaska and brought in to the Seattle Marine Hospital. The knife wound was in one of the extremities, and he came down with some premonitory symptoms of tetanus. A diagnosis of tetanus was made, and an early tracheotomy was done under local anesthesia. He was treated with intravenous and intramuscular injections of the passive serum and was also given massive doses of penicillin and sedation. This patient recovered. Of course, he had a secondary wound closure of the extremity.

The third one was a Norwegian seaman who came in primarily with cerebral symptoms, with headache, pain in his neck, and also cramps in his abdomen. While he was a little queer, fortunately our psychiatrist did not get to him. Instead a diagnosis of tetanus was made. He likewise had a knife wound of an extremity, sutured primarily. We were able first to do a tracheotomy and *en masse* excision of his wound under local anesthesia. He was treated with intravenous and intramuscular serum and massive doses of penicillin. He also recovered. I think this should illustrate the value of early tracheotomy and the value of excising the focus early.

I would like to ask Dr. Nettrour if he does not think that probably at the end of another two weeks, certainly at the end of three weeks from the time of his initial dose of the antiserum in the second case, another dose should have been given.

W. S. NETTROUR (closing): Dr. Carothers stressed the local lesion and the early diagnosis. I think that is most important. I believe, as he does, that careful examination will show up in practically all cases. Also, his point of giving gas gangrene prophylaxis is well recognized, both being anaerobic.

It has been pointed out that some of these cases of tetanus need no treatment, that they will remain a local disease. In some of them the toxin is already fixed, and no amount of treatment will help, but it is this large intermediate group that we are trying to save, the ones in whom early intensive treatment will help.

Dr. Quigley brought out quite a problem in that third group about which he spoke. In other words, if we have tetanus following a hypodermic injection, a bed sore or a needle jab in a baby, how can we immunize people by passive immunization? I wrote that same question to Dr. Magath, in charge of the laboratories at Mayo Clinic, and he said there is no satisfactory answer. In other words, active immunization as is done in the Armed Services is the only answer. The experience of the Navy in the period between the first and second World Wars was that they had only 10 per cent of tetanus cases among those with active immunization, and almost 100 per cent among those with passive immunization. In other words, if you try to judge which ones will get tetanus, you will miss nine out of ten.

I believe Dr. Kuehner's point on tracheotomy is very good. I believe the first man should have had a tracheotomy because he had respiratory stress and because he had a wet chest. At that time, however, the consultant believed that because of

the lack of cyanosis and because of the fact of suction, they could accomplish the same thing. He was carried in comfort by intramuscular nembutal, so there was no question of the comfort of the patient.

Also, Dr. Kuehner's point of the late onset I believe was probably due to the fact that the man did have the serum.

Whether we should give 10,000 units instead of the 1,500—many people recommend 3,000, a double dose—again we have the problem of giving a large group of people serum, the same problem that Dr. Magath pointed to. I do not believe the 10,000 units will solve it. There have been two cases in the Carnegie Library in Pittsburgh who have died. One died from antitetanic serum. The second case died of tetanus following a minor injury. In other words, tetanus toxoid would have saved both of them.

I think Dr. McKim's point of the prevalence of tetanus in diabetic gangrene is very good and should be remembered by all of us. The question of using anesthesia intrathecally I think also is excellent. We have found rather than using the anesthesia, we can obtain the same results and get relaxation by intramuscular barbiturates, and our anesthetist at West Penn Hospital believes there is less risk.

I was very much interested in the three cases that Dr. McGowan presented. The question of eradicating the focus is debatable. It is recommended by many. Others recommend just the local infiltration of the tetanus antitetanic serum there. Most of them believe that it is a circulating toxin as well as the local process. I can see that if the local focus was eradicated, it would be a good thing. Whether surgery will eradicate it in the later stages is doubtful in my own mind.

One thing I would like to stress is the importance of Dr. Kuehner's point of getting industry to immunize these people. The pediatricians are immunizing them, and we think that industry should, too. In most cases the large industries have a nurse, a hypodermic syringe and the patients. All they need is to give the toxoid.

# SELECTIVE PHYSIOLOGIC DÉBRIDEMENT WITH TOPICAL TRYPTIC ENZYMES*

HOWARD G. REISER, M.D., L. C. ROETTIG, M.D. AND G. M. CURTIS, M.D.
*Columbus, Obio*

THE success attending the use of crystalline trypsin for the cavity débridement of pleural empyema[1,2] logically suggested that the course of other fibrinopurulent necrotic lesions might be favorably influenced. Since the first studies, begun in 1950,[3] our personal experience includes more than 2,000 surface applications of crystalline trypsin with an additional 1,000 applications by our colleagues.[4] The rapidity with which trypsin will liquefy fibrin, thick pus and necrotic protein is impressive. Figures 1A and 1B illustrate a characteristic result following a single application of trypsin powder. Despite the good results in our series and in the series of other investigators, a faster and more profound lysis in the débridement of lesions containing collagenous substances, particularly necrotic skin and fascia, seemed desirable. Among the numerous enzymes elaborated by the pancreas, chymotrypsin is abundant and is active in the same environmental conditions as trypsin. The feasibility of the use of this enzyme alone or in combination with trypsin was therefore investigated.

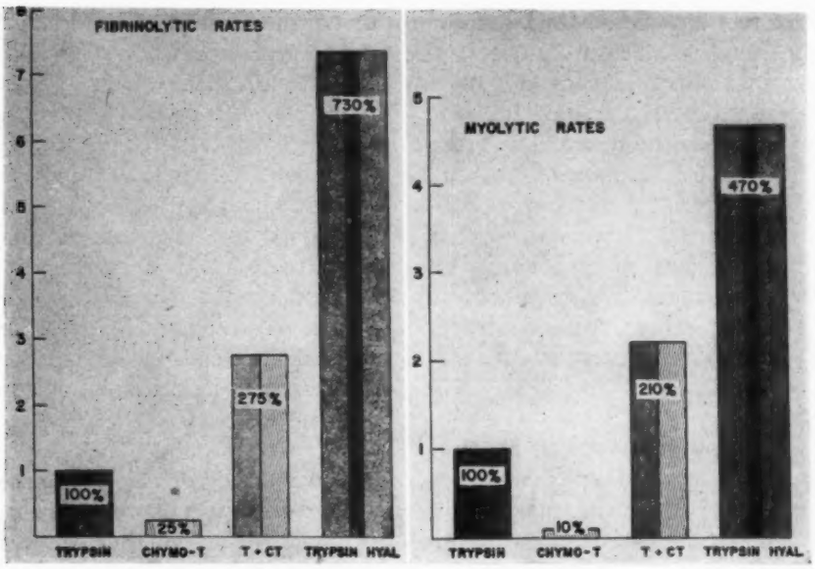
1A 1B

FIG. 1. A, traumatic hand lesion seven days following repair. The effort to preserve tissue resulted in numerous areas of slough which became infected. To this area 100,000 units of trypsin alone were applied as a powder. B, twenty-four hours after the single application of trypsin powder. The entire slough was liquefied leaving a healthy, clean surface.

## PRECLINICAL INVESTIGATION

Preliminary studies demonstrated that chymotrypsin while only moderately effective alone when combined with trypsin in equal enzyme strength increases the rate of proteolysis far above that which might be expected

* From the Department of Surgical Research, Ohio State University, Columbus, O. These studies were assisted by a grant-in-aid and generous supplies of crystalene enzymes—trypsin (tryptar®) and chymotrypsin—by The Armour Laboratories.




2 3

FIG. 2. One per cent fibrinogen clots were subjected to: trypsin 250,000 units; chymotrypsin 250,000 units; trypsin plus chymotrypsin āā 125,000 units; trypsin plus hyaluronidase 250,000 units plus 250,000 units, respectively. The rate of trypsin lysis is taken as the basis for comparison. The factors which influence the startling increase in enzyme lysis in the hyaluronidase series are still under investigation.

FIG. 3. Autopsy muscle in 10 gm. samples was subjected to: trypsin 250,000 units; chymotrypsin 250,000 units; trypsin plus chymotrypsin āā 125,000 units; trypsin plus hyaluronidase, 250,000 units plus 250 units, respectively.

from the additive sums of trypsin and chymotrypsin acting separately. (Figs. 2 and 3.)

From the standpoint of toxicity chymotrypsin is about five times more toxic than trypsin when injected into closed body cavities (pleural and peritoneal spaces). Applied to an open wound, either as a dry powder or a concentrated solution, toxic reaction has not been observed nor has the healing process been altered. Table 1 summarizes wound healing studies in which two symmetric wounds were made on the dorsal cutaneous surface of depilated guinea pigs. In order to secure "identical" lesions the skin was incised with a sharpened 7 mm. circular laboratory corkborer. Two experimental series were established. In the first the experimental wound was treated daily by saline cleansing followed by chymotrypsin powder application. The control lesion was cleansed with saline only. Both lesions were dressed with a sealing patch of adhesive tape. In the second series the experimental wounds were subjected to saturated wet dressings of chymotrypsin dissolved in Sorensen's phosphate buffer at a pH of 7.2. The control lesions were treated with buffer only. A patch of waterproof adhesive sealed the wound and wet dressing.

TABLE 1
DRY CRYSTALS

| Control Wound | | Experimental Wound | |
|---|---|---|---|
| Size (mm.) | Healed (days) | Size (mm.) | Healed (days) |
| 8 | 17 | 8 | 17 |
| 9 | 16 | 8 | 16 |
| 9 | 15 | 7 | 16 |
| 7 | 15 | 8 | 15 |
| Wet Dressing | | | |
| 10 | 11 | 8 | 11 |
| 8 | 15 | 8 | 16 |
| 9 | 14 | 9 | 13 |
| 8 | 10 | 8 | 10 |

In each series a fifth animal was subjected to a larger pair of wounds (20 mm.) and treated similarly. The healing edges were biopsied in

each of the three tertiants when the wounds of the experimental series were approximately 25, 50 and 75 per cent healed. The wounds of the whole series were excised the day following complete gross epithelization. Grossly (Table 1) there is no significant difference in the rate of healing in either series. Microscopically the control wounds are identical with the experimental wounds during and after healing. The normal sequence of events transpire without tendency to excessive scarring, epithelial hypertrophy or inadequate tissue replacement.

## CLINICAL STUDIES

Based on preliminary observations an equal mixture of chymotrypsin and trypsin was used. The proportion is based on enzyme activity rather than weight of substance. In order to secure satisfactory results careful attention must be given to the factors of application.

*Adequate enzyme concentration:* This is achieved by applying the enzyme system as a powder. To this end a powder blower will give a uniform dusting; however, it tends to be wasteful, disbursing the finer crystals into the air. A shaker bottle, when it can be used, wastes a minimum of the enzyme. Care should be taken to contact the *entire* wound area. If the lesion is pocketed, or if the debris is under an extensive tissue flap, it has been found advantageous to incorporate 100,000 to 200,000 units of the enzyme system in a gelatine capsule. The capsule is perforated with a large needle and inserted with the aid of a suitable instrument into the deepest recess of the wound. This latter method modification especially lends itself to the treatment of osteomyelitis, the focus of which frequently lies at the bottom of a sinus tract. Following enzyme powder application a patch of sterile waterproof material is applied *directly* to the wound—rubber sheeting, plastic film, oiled silk and heavy cellophane have all been found satisfactory. Over this may be applied any desired absorbent dressing. This dressing method is essential since with it a layer of enzyme solution is maintained in contact with the lesion rather than being immediately absorbed into a gauze dressing.

*Solution of the enzyme:* It is axiomatic to say that physiologic processes require the proper state of hydration in order to function adequately. This physiologic débridement too must be in a sol phase in order to succeed. Most lesions will supply adequate tissue juice or contain sufficient liquid pus to dissolve the enzyme. However, if the lesion is encrusted or dry, preliminary soaking with Sorensen's phosphate buffer (pH 7.2) is imperative. In the absence of the buffer saline may be used.

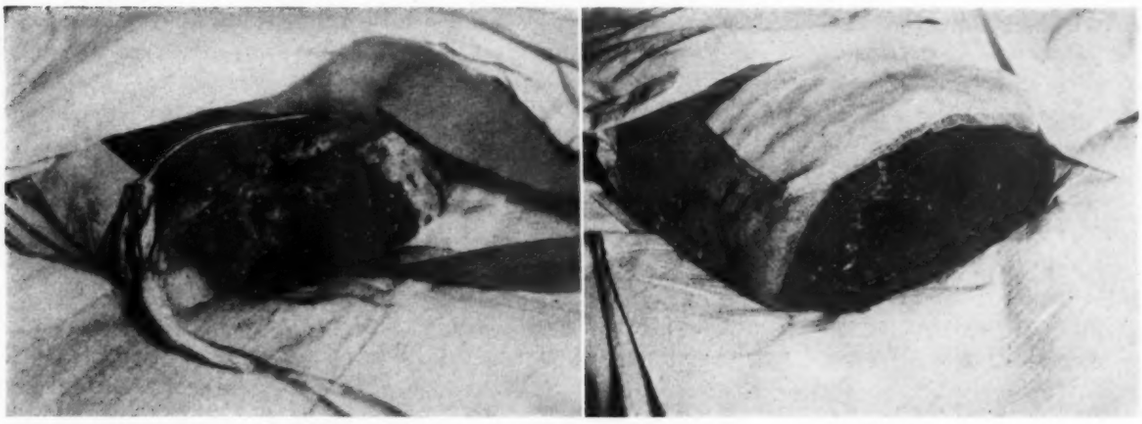
*PH of the lesion:* Ordinarily this is of little concern since most lesions are within the active range of the enzyme—slightly alkaline. In order to aviod disappointment the lesion may be tested with nitrazine paper. Should it be acid, a preliminary wet dressing with buffer is indicated. As treatment progresses it will be found that initially acid lesions will become alkaline.

## RESULTS

The number of applications of this enzyme system exceeds 500. No specific lesion has been selected, but rather the class of lesions presenting a problem in the removal of fibrinopurulent necrotic debris has been utilized. To illustrate the effectiveness of the débridement with this enzyme system a few specific cases will be presented.

*Bilateral Necrosis of the Heads of Metatarsal V.* The lesions resulted from arterial insufficiency in a patient with "controlled" myelogenous leukemia. The cutaneous area overlying the heads of the metatarsal were black and dry. The underlying substance was fluctuant. One of us (H. G. R.) was invited to treat the right-sided lesion while members from another surgical service were asked to treat the left-sided lesion. The right lesion was incised and a capsule containing 100,000 Armour enzyme units, half trypsin and half chymotrypsin, was inserted on each of two successive days. Copious drainage followed. The necrotic soft tissue disappeared leaving a red-freshened surface. On the third day the metatarsal head separated and was removed. On the day following the third capsule of trypsin the wound was completely free of visible debris. However, daily dressing and enzyme powder dusting was continued until the wound healed ten days later. The contralateral lesion treated by incision and drainage, saline soaks and local antibiotics unfortunately progressed ending in mid-leg amputation.

*Comment:* This lesion was quite characteristic of ischemic necrotic lesions which respond quickly to enzymatic débridement. Continued enzyme therapy beyond the débridement period is thought to facilitate healing by keeping the lesion moist, preventing the accumulation



4A 4B

FIG. 4. A, seven days postamputation; note the extensive necrosis on the surface of the stump. B, seven days after insertion of chymotrypsin and trypsin capsules under lateral and medial skin flaps. At this point the stump was closed.

of protein coagula and maintaining the presence of neutrophils. An exudative response abundant in healthy appearing neutrophils is characteristic when trypsin or chymotrypsin is applied to a clean lesion.

*Gas Gangrene.* This infection resulted following a major vascular accident in the left lower extremity in a sixty-four year old white woman with uncontrolled diabetes. Extensive necrosis and knee joint destruction dictated mid-thigh amputation. Despite continuous peroxide irrigation and parenteral antibiotics during the first seven postoperative days tissue destruction progressed. (Fig. 4.) On the eighth day several 250,000 unit capsules of trypsin were placed under the lateral skin flap as high as Poupart's ligament. Another capsule was inserted under the anteromedial skin flap along the adductor canal. The accessible surfaces were dusted with the enzyme. After the fourth daily treatment the entire amputation stump was free of gross debris and the skin flaps began to stick. At this point secondary closure was done and tubular drains were incorporated into the highest recesses of the wound. Into these tubes 100,000 units of the enzyme system dissolved in buffer was inserted daily until the pockets no longer existed. This required an additional seven days. Solid healing resulted and the stump was in excellent condition on the last date of observation, five months after hospital discharge.

*Comment:* It should be pointed out that the entire fascia lata as well as the iliotibial tract was involved. The fascial divisions between muscle groups were similarly affected. Characteristically the enzyme system renders necrotic fascia (as well as necrotic skin) into a glutinous mass through which tough collagen bundles course. If these bundles are severed, large amounts of loosened slough can be removed in each twenty-four-hour period following enzyme application. Needless to say the process is thereby hastened. These dead tissue masses would eventually be liquefied as in the case wherein the slough is inaccessible. Again continued enzyme therapy furnishes a safety factor which keeps the wound clean during healing.

*Decubitus Ulcers.* Numerous decubitus ulcers have been treated. These are most effectively attacked by preliminary buffer wet dressings. Most ulcers will be completely débried in three to seven days. The large extensive ulcers seen in paraplegics require longer periods. With preliminary enzymatic débridement the lesion is ready for grafting, or if closure is feasible, no tissue need be removed. This results, therefore, in considerable tissue salvage since surgical débridement invariably removes considerable viable tissue.

*Other Lesions Treated.* Postradiation, sloughing carcinoma of the breast was treated. This lesion was freed of debris and odor. Healing, of course, did not occur.

There was an infected sloughing cutaneous leukemic infiltration. Total débridement following powder application was obtained in five days. The lesion subsequently healed after x-ray therapy. Infected amputation stumps respond rapidly and can be managed on an outpatient basis.

A retroperitoneal abscess following nephrectomy had failed to close after one month of suppuration, despite saline irrigation, parenteral and local antibiotics. The lesion closed after three weeks of trypsin-chymotrypsin solution instillations.

## OBSERVATIONS

This new enzyme system is vigorously active at the time of application. It is not an intermediate, dependent upon the presence of another substance which may or may not be present in significant quantities in the wound. Continued use of the enzymes has not led to decreased effectiveness nor manifestation of local or general sensitivity. Of further significance is the broad spectrum of non-living protein which the enzymes will attack. Its usefulness is therefore not limited to those lesions in which a specific protein comprises the bulk of the debris.

Following the application of the combined trypsin-chymotrypsin enzymatic débridement, fibrin lysis, pus liquefaction and debris disruption occur in a matter of minutes. The difficult problem is the lysis of collagen bundles so numerous in connective tissue, fascia, tendons and skin. Trypsin alone is effective in repeated applications. However, with the addition of chymotrypsin to the débridement mixture the collagen bundles are rapidly softened and may be lifted from the viable, underlying tissue. Necrotic fascia and skin become a glutinous mass from which may be expressed considerable liquefied protein, leaving behind a rough, stranded network. It is the network which requires severance if speed in débridement is desired. Complete cleansing rarely requires more than two weeks of therapy, most lesions responding readily in five to eight days.

The extension of enzymatic therapy beyond the point of complete débridement is based on five observations: (1) The elimination of the surface coagulum which tends to form before it is completely covered by vigorous granulation tissue. (2) Continuing stimulation to the wound to produce an exudate abundant in neutrophils. Smears of the exudate following enzyme powder invariably manifest numerous intact leukocytes.[1,3] Observations on polymorphonuclear leukocytes displaying increased motility and phagocytic power when treated with tryptic enzymes are being evaluated.[5] However, it seems logical to conclude that the continued presence of fresh, vigorous neutrophils in an open wound is highly desirable from the standpoint of bacteriologic defense. (3) Antibiotics display increased potency in preventing bacterial growth.[6] This phase of tryptic activity is being extensively investigated. (4) Some bacteria cannot grow in the presence of tryptic enzymes while the physiology of others is so altered that they do not tolerate minor noxious changes in their environment.[6] (5) Continued application of tryptic enzymes does not delay or alter the healing process.

For these reasons we continue therapy until complete firm healing or epithelization occurs, and the clinical results have been rewarding.

## SUMMARY

1. The author's experiences with crystalline-trypsin (tryptar) has been briefly reviewed.

2. Chymotrypsin and trypsin together in equal enzyme strength exhibit potentiated proteolysis—more than 200 per cent more rapid than an equal concentration of trypsin alone.

3. Chymotrypsin is relatively non-toxic, does not delay healing or alter the microscopically visible course of events when applied to open wounds.

4. A number of illustrative cases show the adequacy of the new enzyme system of trypsin and chymotrypsin in handling fibrinopurulent necrotic lesions which have failed to respond to the usual surgical measures.

5. The combination of trypsin and chymotrypsin in equal enzyme strength when properly applied to the fibrinopurulent necrotic lesion which has failed to respond adequately to the usual surgical measures, rapidly eliminates noxious elements which prevent healing. In the presurgical preparation of the lesion in which tissue salvage is imperative this new enzyme system offers a safe, effective means of eliminating all necrotic detritus, leaving the healthy viable tissue intact and unaltered. Herein lies the uniqueness of this physiologic enzymatic cleansing process: it automatically halts its débridement at the fine line between life and death.

## REFERENCES

1. Reiser, H. G., Roettig, L. C. and Curtis, G. M. Tryptic Debridement of Fibrinopurulent Empyema, pp. 17–24. Surgical Forums. Philadelphia, Pa., W. B. Saunders Co.

2. Roettig, L. C., Resier, H. G., Habeeb, W. J. and Mark, L. The use of trypsin in chest disease. *Dis. Chest*, 21: 245, 1952.
3. Reiser, H. G., Patton, R. and Roettig, L. C. Tryptic debridement of necrotic tissue. *Arch. Surg.*, 63: 568–575, 1951.
4. Conners, A. C. et al. Trypsin in osteomyelitis. To be published.
5. Reiser, H. G. Unpublished data.
6. Birkland, J. and Englehart, W. Department of Bacteriology, Ohio State University. Personal communication.

DISCUSSION

L. C. Roettig (Columbus, O.): First, I want to thank you for this opportunity which you have given Dr. Reiser and myself to present our paper before the Association, because I believe this is where the material really has a place. I would like to emphasize one point, which I am sure Dr. Reiser would want to emphasize, and that is that many of the papers which have been written on enzymatic débridement have failed to stress the point that the investigators believe is probably the most important place that enzymatic débridement has in our treatment of traumatic injury, and that is its tissue-sparing action.

Very frequently we will see massively contused traumatic wounds of the extremities or of the soft tissues, and the question comes up, can these wounds be débrided and save the extremity? This is the place where physiologic débridement really fits in because, as Dr. Reiser emphasized, this method does stop at the fine line between life and death. By the use of enzymatic débridement on these extensive wounds you can actually spare and save the viable tissue, thereby on many occasions saving an amputation or a tremendous loss of soft tissue to the patient.

We are also working on burns, and I think that it has a very definite place in burn therapy, but this is going to take a great deal more work than we have done at the present time.

Howard G. Reiser (closing): The place that tryptic enzymes might occupy I think can be pointed out by three salient features.

One, which I have already mentioned, is the immediate and rapid removal of the debris.

The second one, which Dr. Roettig has brought out, is the selectiveness of this process. Only the necrotic tissue is removed. Even the tiniest island, indeed a single living cell, will be spared by tryptic débridement. Much of this important tissue, would certainly reach the specimen pan unseen by the knife, or in the presence of continuing infection would certainly succumb in the melee of continued bacterial action.

Third, is the characteristic exudate following the first and all subsequent applications of tryptic enzymes. These exudates characteristically abound in polymorphonuclear leukocytes which are healthy in appearance. It would appear that a chronic wound is converted to a limited acute inflammatory phase, ready to heal and eager to combat further infection.

Selective physiologic débridement with topical tryptic enzymes offers the surgeon a cogent weapon whereby he can attack the fibrinopurulent necrotic lesion, which is a problem, with greater expectancy of reward from his surgical skills and judgment.

# THE PLASMA SUBSTITUTES*

## PROGRESS REPORT

BRIG. GEN. SAM F. SEELEY, M.C. AND LIEUT. COL. EDWIN J. PULASKI, M.C.
*Washington, D. C.*

EFFECTIVE blood volume loss is the primary mechanism of shock in military and civilian disasters. The most common initiating cause of volume loss is hemorrhage, while mixed losses from the crush syndrome and those from burns and infections, which are predominantly plasma, are among the other frequent causes. In most cases, if the blood volume is promptly restored to normal, a reversal of most or all of the deleterious effects of shock will follow.

The supportive effects of fresh whole blood are remarkably superior to any other available mixture. Of importance are the red blood corpuscles, not only in regard to oxygen transport and buffering capacities, but also because of the space-occupying function. The oncotic pressure exerted by the plasma proteins is also an important property of blood. However, plasma and other blood fractions are less effective resuscitative agents because they lack erythrocytes. They are also less efficient than whole blood in that a significant portion of the proteins is rapidly lost into the extracellular compartment with only a transient increase in blood volume in response to infusion. Indeed, the beneficial effects are often of no more than three hours' duration.

The problem of maintenance of an adequate supply of whole blood and its derivatives for use in a national emergency has led to investigation of plasma substitutes. The function of a practical plasma substitute must be to sustain the circulation by restoring blood volume, in whole or in part, for a period sufficient to allow further spontaneous recovery from shock or until more definite treatment can be administered.

The important criteria for an ideal plasma substitute are as follows: (1) should retain within the circulation at least half of the administered fluid volume for at least twelve hours; (2) should have an oncotic pressure equal to that of plasma; (3) should have a viscosity compatible with intravenous injection at all environmental temperatures; (4) must be non-toxic, having no immediate adverse effects and causing no organic derangement, either immediate or delayed; (5) should be ultimately excreted or metabolized; (6) must be non-antigenic; (7) must be easily and completely sterilized; (8) must be stable in storage for long periods; (9) must be capable of mass production with a constant composition in order to meet the economic and logistic requirements of the present national situation.

The significance of molecular size of the different plasma substitutes should be mentioned. All of the substitutes available contain molecules of widely varying size during manufacture. During processing of the product for clinical use, the largest and smallest molecules are discarded, leaving mainly those in the 30,000 to 100,000 molecular weight range. When the resultant product is infused, most of the material can be accounted for in the blood. The kidney is the only significant route of excretion; only trace amounts can be recovered in the bile, feces and expired air. The smaller molecules are not retained in the vascular tree but are rapidly removed from the circulation and the body. The small molecules, therefore, have only a transient clinical effect. The larger molecules do not rapidly permeate the capillary walls; part remain in the circulation and part accumulate in the interstitial fluid spaces. Effective retention of molecules results in hemodilution, with a decrease in hematocrit, erythrocyte count and total protein concentration. That storage in the body of very large molecules of plasma expanders does occur has now been demonstrated. A far more serious disadvantage of the presence of molecules larger than 100,000 is their interference with typing and cross-matching of blood and increased viscosity and sedimentation rate. For these reasons molecules with a molecular weight larger than 100,000 must be excluded, if

* From the Surgical Service, Walter Reed Army Hospital, Washington, D. C.

possible, from products designed for clinical use.

Several plasma substitutes were investigated after the introduction of acacia during World War I; others are now under active study. At the present time three of these appear to meet most of the aforementioned criteria of acceptability. These are dextran, polyvinylpyrrolidone and the fluid gelatins. Their current status will be briefly summarized.

### DEXTRAN

The dextrans are an ill defined group of macromolecular carbohydrates produced from sucrose by a specific group of bacteria, especially of the genus leuconostoc. The chemical nature and the molecular form, size and shape of the dextrans have been well studied. Methods are available for determining the range and distribution of molecular sizes in any given sample.[2,3]

Dextran available in this country is produced by selected strains of leuconostoc. The conditions of growth and preparation are such that the product supplied has the desired colloid osmotic pressure and viscosity in a 6 per cent solution.* It has now been amply demonstrated that dextran restores circulating blood volume, maintains blood pressure and promotes survival in experimental as well as in human shock. The question of toxicity of clinical dextrans cannot be considered settled until more is known about the fate of infused dextran. The small molecules are rapidly excreted in the urine. At least part of the retained dextran is metabolized. Michie[4] and his collaborators find no clear evidence of renal damage, although in some cases a depression of tubular function ($TM_{pah}$) following the administration of dextran has been noted. The latter phenomenon was not seen in the three cases reported by Fleming et al.[5] Reinhold[6] has studied liver function after the postoperative administration of dextran. He found only effects referable to anesthesia and operation, with the exception of a persistent bromsulfalein retention in one case.

One of the unsolved problems concerning dextran is that of its antigenicity. In spite of satisfactory experience with Swedish dextran in Europe investigators and surgeons using the same material in this country, especially in military personnel, found an alarmingly high incidence of reactions. Intensive search for the mechanism has only partially elucidated the immunologic phenomena involved. A careful study by Kabat, Warren and others[7] showed that adverse systemic reactions are definitely less frequent with the domestic than with the Swedish preparation. A group of seventy young men were given 500 ml. of a clinical dextran preparation having a grand average molecular weight of 59,000 and an average of 175,000 for the upper 11 per cent of the weight distribution. Only ten patients showed any reaction, none classified as severe or moderate. Of the ten mild reactions, one had two hives, and four had only a single hive each. A parallel test, using dextran having more large molecules, produced many more reactions, and it appears that molecular size and shape without regard to specific immunologic properties may be one cause of reactions. Although they found a high incidence of positive skin reactions to leuconostoc protein in individuals who had never received dextran, there was no correlation of positive skin reaction with adverse systemic reactions to intravenous doses. They also failed to find true sensitivity in individuals tested from four to twenty-three months after an infusion of dextran. Warren[8] has shown that with certain strains of leuconostic there is a correlation between the precipitin titer and reactions occurring in patients infused with dextran. He also found a significantly frequent appearance of leuconostoc strain-specific agglutinins in the unexposed population, but their presence cannot be correlated with individual histories of immunizations. In about 50 per cent of individuals the administration of dextran produces a rise in titer of agglutinins. In effect leuconostoc strains used to produce American clinical dextran are least likely to produce troublesome reactions. Ricketts[9] has recently found a precipitation reaction between dextran and fibrinogen. He suggests the possibility that the uptake of a dextran-fibrinogen compound by cells of the reticuloendothelial system might account for the demonstrated storage of dextran in organs especially well supplied with those cells.

### POLYVINYLPYRROLIDONE

Polyvinylpyrrolidone, or PVP, is a synthetic macromolecular polymer produced in Germany early in the war and quite widely used there as a

* Trade names of some of the current preparations are: Macrodex, Expandex, Intradex and Plavolex.

plasma expander. Unfortunately, records of experience on which sound conclusions can be based are not obtainable. PVP is entirely synthetic, the raw materials being ammonia, acetylene and formaldehyde. It is freely soluble in saline solutions and the 3.5 per cent solution has a colloid osmotic pressure and viscosity of the desired values. Preparations* free of very large molecules do not seriously interfere with cross-matching, although it is still recommended that cross-matching be done, if possible, with the recipient's blood drawn before *any* plasma expander is given. The fate of injected PVP in man is still unknown, but deposition and storage of a portion of any dose, probably the larger molecules, does occur and evidence of any deleterious effects is being sought for in several laboratories. There is no evidence that PVP is metabolized. PVP is non-antigenic, except in the dog, a fact which makes the animal of somewhat doubtful use for investigation purposes.

The use of PVP is widespread in France at the present time.[10] The reported reaction rate, 0.5 per cent, is as low as with any intravenous therapy. Cournand and his collaborators[11] infused 1 L. of PVP into each of fifteen male patients without shock or circulatory failure. The plasma fluid increment was only 300 to 400 ml. at the end of twenty-four hours, at which time 40 per cent of the PVP had appeared in the urine. At the end of seventy-two hours 28 per cent of the dose was unaccounted for in blood and urine; it may, therefore, have entered some unknown storage depot in the body. Zollinger and his group[12] found satisfactory initial reponses to PVP in thirty-nine of forty-two patients in shock from hemorrhage, trauma, burns or other causes. Furthermore they did not find harmful effects as evidenced by bone marrow, biopsy and autopsy tissue studies in patients followed as long as fourteen months. At the present time additional information is needed on (1) what constitutes the optimal molecular fraction for clinical use; (2) the degree of effectiveness of each fraction in sustaining blood volume.

## GELATIN

Gelatin preparations have been used for several years and their value and applicability are widely recognized. Ordinary gelatin preparations are heat-sensitive and both colloid osmotic pressure and viscosity are reduced by autoclaving. As with dextran and PVP the molecules of weight below 30,000 are not retained in the blood stream but are excreted by the kidneys. Gelatin in the blood stream acts like plasma, is excreted without toxic effects and has been found satisfactory in the treatment of wound and burn shock. The A.M.A. Council-approved gelatin preparations are nontoxic and non-antigenic and can be completely sterilized.

Chemical modification of the gelatin molecule gives promise of success in overcoming one serious drawback, namely, the tendency to gel at low temperatures. This tendency greatly limits its usefulness under field conditions. Oxypolygelatin and fluid gelatin, with gel points near freezing, are being tested to determine their acceptability as plasma expanders. Samples of fluid gelatin, gelling at 32 to 38°F., have been tested by Vars[14] in dogs and in man with very promising results.

## COMMENTS

Clinical experience with plasma expanders is well summarized in a comprehensive review by Gropper et al.,[15] which the authors have been privileged to read prior to publication. It appears that plasma expanders find greatest utility in cases in which the blood loss does not exceed 20 per cent of the normal blood volume. In such cases saline may also be given as a useful supplement. If from 20 to 35 per cent of the blood is lost, it is inadvisable to dilute the plasma colloids further; in this case the expander should be used without saline. Blood loss of over 35 per cent of the normal volume produces a deficit in red blood corpuscles sufficient to demand the use of whole blood. The judicious use of expanders can be of supplemental value.

Much more additional information is needed before the use of plasma expanders can be considered in the routine therapy for shock. Current developments in manufacture are resulting in improved products which must nevertheless be subjected to careful test. During testing a complete record of every use should be made. This should include not only the immediate effects on the patient but also the long-term observations. Records of the effects of infusions on blood pressure are

* Trade names of some of the current preparations: Periston, Macrose, Subtosan, Plasmosan, and Plasdone.

of course of prime practical importance. Measurements of blood volume are of equal interest, but it must not be forgotten that the current methods for measuring blood volume are of doubtful validity in patients in profound shock.

The ideal plasma expander has not yet been produced. If it is found, it may bear no relation to those which have been discussed. Therefore, it is essential that each suggested expander be critically tested and the results be fully published, so that the knowledge thus gained may be usefully applied to the search for a practical plasma substitute.

REFERENCES


1. Strumia, M. M. and Sample, A. B. Modified globin. I. Method for preparation from human erythrocytes. *J. Lab. & Clin. Med.*, 37: 959–968, 1951.
2. National Bureau of Standards Reports, 1028, 1160, and 1713: Molecular properties of plasma substitutes, 1951, 1952. Restricted.
3. Report of a Working Conference on Dextran. Peoria, Ill., Northern Regional Research Laboratories, U. S. Dept. Agriculture, (July 9) 1951. Restricted.
4. Michie, A. J. and Ragni, M. C. The effects of dextran on kidney function, Reports to the Subcommittee on Shock. National Research Council, July and November, 1951.
5. Fleming, J. W., Cargill, W. H. and Bloom, W. L. Effects of intravenous administration of dextran on renal function (19460). *Proc. Soc. Exper. Biol. & Med.*, 79: 604–606, 1952.
6. Reinhold, J. G. and von Frijtag Drabbe, C. A. J. The influence of plasma expanders on the liver as shown by certain tests of liver function. Report to the Subcommittee on Shock. National Research Council, November, 1951.
7. Kabat, E. A. et al. Report on project for evaluating effects of Army immunization and for determining any correlation between cutaneous sensitivity to dextran and the incidence of adverse systemic reactions to infusion of dextran, I and II. Reports to Subcommittee on Shock. National Research Council, January and April, 1952. Restricted.
8. Warren, J., Fedes, J. C. and Kent, J. F. Serological reactions of human sera and rabbit sera with clinical dextran. *Bact. Proc.*, p. 93, 1952.
9. Ricketts, C. R. Interaction of dextran and fibrinogen. *Nature*, 169: 970, 1952.
10. Eirich, F. R. Memo to National Research Council, August 9, 1951.
11. Bader, R. A., Kim, S. W. and Cournand, A. Clinical use of PVP. Report to Subcommittee on Shock. National Research Council, January 21, 1952. Restricted.
12. Zollinger, R. M. et al. An evaluation of the synthetic plasma expander polyvinyl pyrrolidone. Report to Subcommittee on Shock. National Research Council, September 17, 1951. Restricted.
13. Parkins, W. M., Perlmutt, J. H. and Vars, H. M. Dextran modified fluid gelatin and other plasma volume expanders in hemorrhaged dogs. *Fed. Proc.*, 2: 116, 1952.
14. Barker, H. G., Elder, J. D., Walker, J. M. and Vars, H. M. The retention of certain plasma volume expanders within the circulation of human subjects following a 1,000 cubic centimeter hemorrhage. *Surgery*, 32: 299, 1952.
15. Gropper, A. L., Raisz, L. G. and Amspacher, W. H. Plasma expanders: a collective review. *Surg., Gynec. & Obst.*, 95: 521–542, 1952.
16. Baer, K. A. Plasma substitutes, except those derived from human blood. 1940–1951. Washington, 1951. Army Medical Library.
17. Scudder, J. Shock syndrome. *Ann. New York Acad. Sc.*, 55: 345–542, 1952.

# A MIDDLE COURSE IN BLOOD AND FLUID REPLACEMENT IN TRAUMA*

JOHN PAUL NORTH, M.D.
*McKinney, Texas*

INSTANCES of resuscitation and recovery from apparent death by trauma are sufficiently rare in medical annals to warrant inquiry into the manner in which they occur. Surgeons have little control over some aspects of severe trauma. In one respect we may often determine the outcome by the decisions we make concerning the giving or withholding of blood and fluids, as the following case illustrates:

On January 21, 1950, a thirty year old Choctaw Indian was shot through the upper abdomen with a .22 caliber bullet while attending a tribal pow-wow at Broken Bow, Oklahoma. This diversion appears not to have interrupted the proceedings, for the victim was pushed out of sight behind the door of the council room as the pow-wow continued. At adjournment four hours later it was assumed he was dead and an undertaker was summoned to take care of the body. In the course of his preparations for the embalming he was startled to detect signs of life. Understandably disturbed, he hurriedly placed the body in an ambulance and transported it 165 miles to the Veterans Hospital at McKinney, arriving ten hours after the shooting.

The patient was in severe shock on arrival, with a blood pressure of 70/50 and a pulse rate of 160. There was an entrance wound at the lower edge of the tenth rib in the right anterior axillary line. The missile itself was palpable beneath the skin of the opposite flank. The tract of the bullet was below the usual limits of the pleura and there was neither physical nor roentgenologic evidence of pleural penetration.

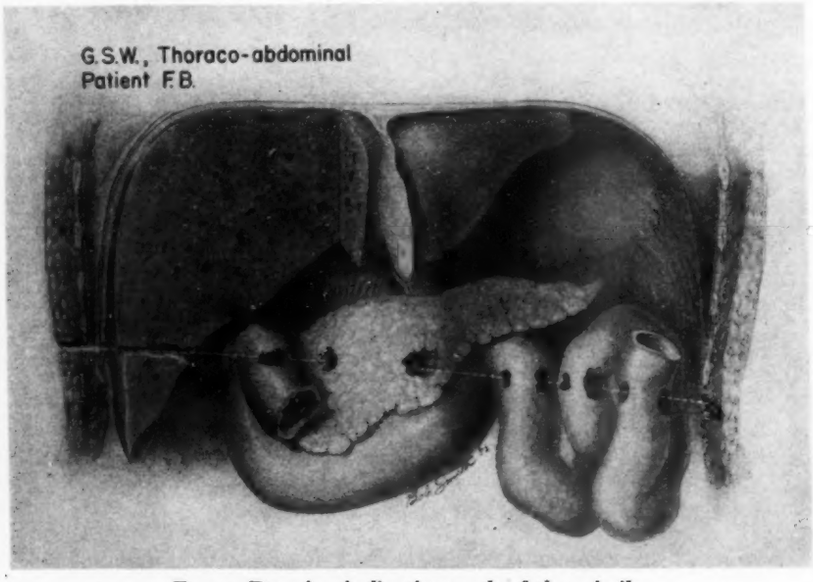



FIG. 1. Drawing indicating path of the missile.

It required 2 L. of blood and 6 L. of Ringer's lactate solution over a period of seven hours before the blood pressure had climbed to 100/80 and the necessary laparotomy could be begun. As the right lobe of the liver was displaced, a sucking wound of the diaphragm was created. It was then evident (Fig. 1) that the missile had crossed the pleural cavity, entered the abdomen and perforated the right lobe of the liver. It had made a ragged tangential tear in the juxtapyloric duodenum and drilled a hole admitting two fingers through the body of the pancreas. It had then coursed through

* Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions of the author are the result of his own study and do not necessarily reflect the opinions or policy of the Veterans Administration.

three contiguous loops of jejunum before finally lodging in the parietes. The repair of the injured viscera was tedious and had to be interrupted from time to time for restorative measures as the blood pressure hovered in the vicinity of 50 mm. systolic. Nevertheless, at the completion of the operation in the early hours of the morning the blood pressure had steadied at 100/80 and the pulse rate at about 120.

During the first twenty-four hours after operation there were two critical episodes, with the blood pressure falling to shock levels. The first was corrected by rapid infusion of Ringer's lactate solution. When shock recurred once more, laboratory data were available to indicate that replacement of both blood and salt deficiencies had already been accomplished. It was evident, however, that additional fluid of some sort would have to be infused if the patient was to be saved. Glucose in water had been ineffective. Plasma proved equally ineffective. (The plasma expanders were not available to us at the time.) Accordingly, it was decided to give more blood, and 500 cc. were transfused slowly throughout the night. The response to this transfusion was dramatic and thereafter the shock problem was under control.

At the close of the first day there were no signs of overhydration despite the large quantities—a total of 11 L.—of fluids which had been given. Normal skin turgor had returned but there was no perceptible edema and the superficial veins were still collapsed.

The patient was not at this time exhibiting any respiratory distress but the following morning he had marked dyspnea. His right chest was flat to percussion and there were audible rales. The x-ray showed two-thirds of the right lung field obscured by effusion, both diaphragms elevated, mediastinal structures shifted to the left and a mottled haziness in the visible lung fields. These findings were interpreted as indicating hemopneumothorax and traumatic wet lung. Considerable relief from dyspnea was afforded at once by the withdrawal of 800 cc. of sanguineous fluid from the right chest. On the succeeding day 1,100 cc. were recovered and profuse secretions were also aspirated from the lungs by bronchoscopy. Decreasing amounts of effusion fluid were removed for two or three more days and pneumonitis remained. One month after injury decortication was done to obtain full reexpansion of the lung. (Figs. 2 and 3.)

During the otherwise critical first twenty-four hours approximately 1 L. of urine was passed. In the following days the urine output ranged from as little as 150 cc. to a maximum of 500 cc. Meanwhile blood urea nitrogen levels rose progressively to a peak of 185 mg. per cent. This "renal crush" syndrome was managed by rigidly restricting the daily intake of fluid to the amount of urine secreted each day plus an allowance of 1,000 cc. to cover insensible losses. Finally on the twelfth day the renal block was broken and diuresis occurred. By following this simple regimen recommended by Muirhead and Hill[5] a mounting azotemia need not be alarming and drastic measures such as the use of the artificial kidney or peritoneal lavage may be avoided.

The sequel to this case history is that soon after this patient had returned to his reservation another Choctaw who admitted to the earlier shooting presented himself at our hospital with a stab wound of the abdomen.

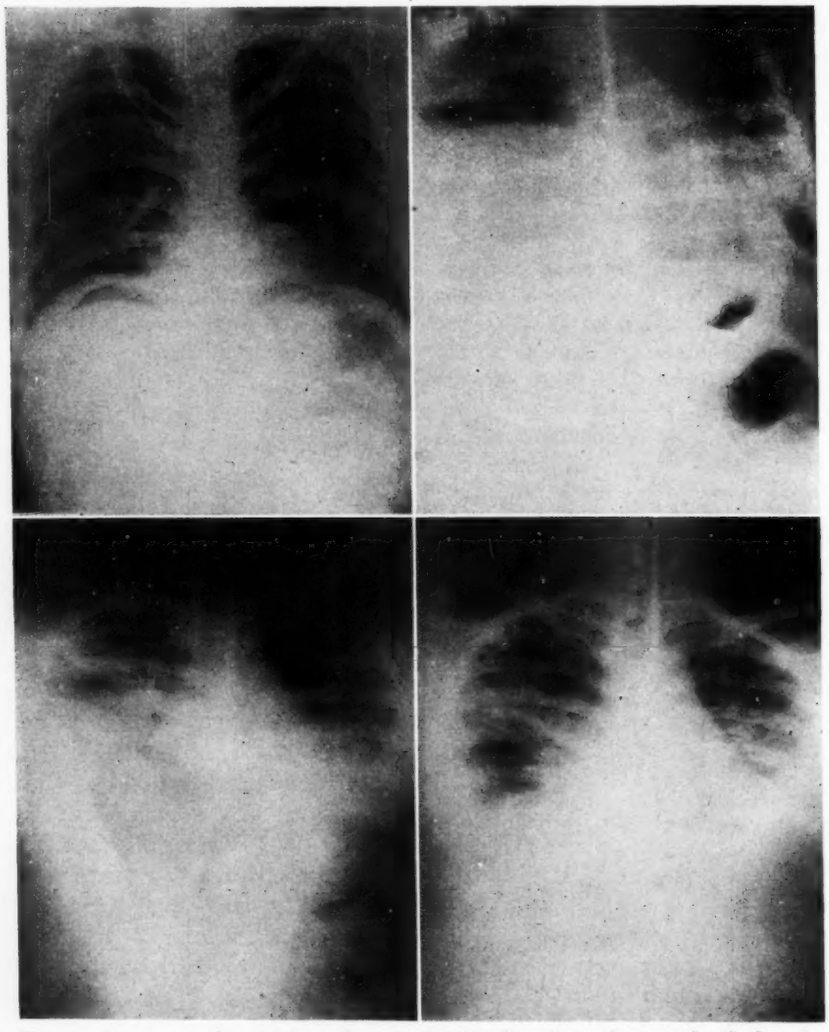
## COMMENTS

No one believed that this patient would recover. Surgeons commonly analyze their cases which terminate fatally but this case presents the unusual opportunity to study the fortuitous chain of circumstances by which one patient survived. What determined that this Indian should cheat the mortician to whom he was initially assigned?

The case involved four distinct phases, namely, primary shock, secondary postoperative shock, traumatic wet lung and posttraumatic renal block. For the sake of clarity they have been described as if consecutive. In reality they overlapped one upon another. In each phase, however, there was only a narrow margin between inadequate fluid replacement on the one hand and overload on the other. Moreover, at nearly every stage what was necessary and proper therapy from one point of view could be deleterious from another.

Admittedly the missile was of small caliber. Nevertheless, it traversed a series of important viscera and initiated a severe necrotizing infection within the abdomen. The callous indifference of the tribe during the early hours when bile, duodenal and pancreatic secretions were extravasating into this man's tissues may in the long run have been beneficial. Certainly in this case there was no blundering interference with the body's natural reactions against trauma. His primary shock was literally treated by leaving him where he lay and postponing for five hours the secondary trauma of transportation. Meanwhile extracellular fluid, mobilized from uninjured areas, diluted the concentrated secretions released within the peritoneal cavity. Fortunately during this period blood loss was not of exsanguinating proportions although doubtless considerable.

One speaks glibly of "irreversible shock" al-



2A 2B

3A 3B

FIG. 2. A, x-ray prior to operative repair showing clear chest and subphrenic collection of gas. B, appearance of the chest thirty-six hours later (bedside film).

FIG. 3. A, x-ray of chest on third postoperative day after removal of 800 cc. of fluid; B, again on the fourth day after additional thoracentesis and bronchoscopic aspiration.

though unable to define precisely when it develops. In this instance obviously the point of no return had not been reached in ten hours after injury. Resuscitation was accomplished in the main by the guidance of changing clinical signs rather than by laboratory determinations. Under circumstances such as these the volume of blood already lost can only be guessed at. Entirely erroneous conclusions can be drawn from hematocrit levels when there is hemoconcentration. Determinations of plasma volume are beyond the capacities of the usual hospital laboratory, especially at midnight. It was therefore assumed that from 3 to 4 units of blood would be required to replenish blood already lost and that perhaps more might be required during the contemplated surgery. There was obviously also a severe deficit of extracellular fluid from displacement of fluid to the traumatized area.[3] The clinical manifestations of blood and salt deficiencies are in general indistinguishable and their correction must therefore proceed simultaneously.[4] In this case the effectiveness of restoration was measured by the return of skin turgor, increasing awareness of the environment, rising blood pressure, declining pulse rate, improvement in the quality of peripheral pulses and warming of

the extremities. These were the main guideposts to shock therapy which dictated that 2 L. of blood, 6 L. of lactated Ringer's solution and seven hours of continuous observation were required before the surgeon lifted his scalpel. These are large volumes of fluid indeed, but not until they had been given did the signs of shock disappear. The necessary operative repair could then be carried out under the best possible circumstances.

In the early postoperative hours the problem was one of supporting blood pressure which showed a tendency to wane despite continuous infusion of glucose and water. Blood chemistry reports were available at this time to aid in the choice of appropriate repair solutions. The plasma sodium concentration was within normal limits. (Plasma $CO_2$ of 12 mEq./L. plus plasma chlorides of 120 mEq./L. equalled an estimated sodium concentration of 132 mEq./L.) The moderate acidosis appeared to be compensated. A deficiency in plasma calcium due to action of pancreatic secretions was readily corrected by calcium chloride intravenously. A hemoglobin of 18 gm. after correction of hemoconcentration did not imply any need for additional transfusion. We had, moreover, been sensitized to the danger of overtransfusion by an experience with a somewhat similar case of duodenal rupture. In that instance a comparable shock-like state after operation had been countered by continued administration of blood even as in this case after normal red cell concentration had already been regained. This resulted in quick death, with autopsy findings of intense hyperemia and hemorrhage in all the viscera. At the moment the dangers of overtransfusion appeared more serious to us than those of overloading with fluids; hence the reluctance to give more blood. There is sound experimental evidence that buffered salt solution is as effective in such a situation as plasma, and is definitely superior to ordinary sodium chloride or glucose solutions.[6] This proved to be true in the first crisis but later blood was required to stabilize the situation. Did the favorable response to blood and to blood alone imply that the sustained postoperative shock was due to inadequate replacement even though the red cell count was normal? How under the circumstances can one differentiate between a blood deficit and salt depletion? Sometimes only trial and error can solve such a dilemma.

How much did the incipient but as yet unrecognized pleuropulmonary complication contribute to this postoperative shock picture? As often happens, the most dramatic feature of multiple injury claims the concentration of the surgeon to the exclusion of others. In this instance on the first day the thoracic component of the wound produced no obvious symptoms and received little attention. The next day, however, the full-blown picture of pneumohemothorax combined with traumatic wet lung appeared, as described by Burford and Burbank.[1] Granted that the perforating wound of chest wall and pleura alone might account for this reaction, may not the large volume of intravenous fluids have played a part in its production? This suspicion is confirmed by the delayed appearance of the wet lung and also by the fact that its usual precursor, chest pain with its involuntary restriction of respiratory movements, was not prominent. Compression of the lung by effusion, mediastinal displacement and obstruction of airways by secretions certainly results in disturbance of cardiopulmonary equilibrium and anoxia. In retrospect it is clear that a water-sealed drain should have been introduced into the right pleural cavity at the time of laparotomy, and it is also conceivable that thoracentesis on the first day might have been even more beneficial than the administration of additional fluid in stabilizing the faltering blood pressure. The production of pulmonary edema by overhydration in the presence of a failing right heart is commonly recognized as well as the dangers of overtransfusion in such patients.[2] This case indicates that even in patients with normal cardiac capacity the indiscriminate administration of fluids may lead to complications although by a different mechanism.

## CONCLUSION

Success with blood and fluid therapy in severe trauma involves steering a course between the Scylla of inadequate replacement and the Charybdis of overload. This middle course cannot be charted in advance. It is not too well buoyed with reliable clinical signs, nor is it clearly lighted by dependable laboratory data. The navigator needs a comprehension of the underlying physiologic and chemical disturbances in their entirety and he must be alert to interpret any variations in the patient's condition. Above all he must not be diverted from

standing a ceaseless watch at the bedside to make the changes in course which may be required from hour to hour. I humbly acknowledge the faithfulness of my resident who was at the helm during the perilous voyage described.

REFERENCES

1. Burford, T. H. and Burbank, B. Traumatic wet lung. *J. Thoracic Surg.*, 14: 415, 1945.
2. Holden, W. D., Cole, J. W. and Portman, A. F. Myocardial intolerance to excessive blood transfusion. *Surg., Gynec. & Obst.*, 90: 455, 1950.
3. Holmes, J. H. and Painter, E. E. Role of extracellular fluid in traumatic shock in dogs. *Am. J. Physiol.*, 148: 201, 1947.
4. Moyer, C. A. Fluid Balance. Chicago, 1952. Year Book Publishing Co.
5. Muirhead, E. E. and Hill, J. M. Treatment of acute renal insufficiency. *Surg., Gynec. & Obst.*, 87: 445, 1948.
6. Rosenthal, S. M. Experimental chemotherapy of burns and shock; effects of systemic therapy on early mortality. *Pub. Health Rep.*, 58: 513, 1943.

DISCUSSION OF PAPERS BY DRS. SEELEY AND PULASKI, AND NORTH

William T. Fitts, Jr. (Philadelphia, Pa.): The Department of Surgery of the University of Pennsylvania has been greatly interested in the problem of plasma expanders for the last several years. A group of investigators headed by Dr. Ravdin has studied these expanders at our institution. I would like to summarize Dr. Ravdin's conclusions to date.

It is important to test these expanders in the hypovolemic state as well as in a patient with a normal blood volume. In patients in the hypovolemic state these expanders remain in circulation longer, and of course it is likely to be in the hypovolemic state that they will be used. In a large number of patients who have been given PVP and have been followed up closely for a period up to seven months, when kidney and liver function tests were performed no evidence of damage to the liver or kidney was found. In some of our patients dextran has given transient kidney damage as shown by kidney function tests of tubular function. These persons have generally not been in a state of shock. It is entirely possible that in a patient with shock who has been given dextran this tubular damage may be sufficient to cause a permanent effect on the kidney.

The gelatins that were used during World War II were not in a fluid state at ordinary temperatures. However, the oxypolygelatins, which we have used recently, have been liquid at room temperatures, have been effective and have shown no deleterious effects.

A word about Dr. North's paper, which I enjoyed very much. Certainly when the injured patient comes to the hospital we do not know how much whole blood he has lost and how much to replace. We do have, however, a quick method of determining approximately how much blood the patient has lost at the operating table and can steer the middle course during operation. This is by the use of dry sponges and then weighing the sponges during surgery. We have replaced as much as 6 or 7 L. of blood at operation in very large thoracic or abdominal procedures and believed that the person left the table with approximately his normal blood volume, the giving of blood having been guided by the dry sponge-weighing technic.

John Scudder (New York, N. Y.): At Presbyterian Hospital we have on hand albumin, gelatin and PVP-macrose in the emergency treatment of decrease in blood volume associated with various types of shock.*

*Albumin.* Colonel Pulaski referred to albumin: Figure 1 shows the electrophoretic pattern of plasma and of its albumin component. Each gram of administered albumin will immediately pull into the circulation 18.5 gm. of water.

Albumin is the safest, most effective plasma volume expander known today. However, it is in short supply and is too expensive.

At Harvard, medical students bled of 1,000 cc. by Dr. Gibson were divided into three groups: untreated controls, those treated with saline and those with albumin. The action of saline on restoration of blood volume was transient while that of albumin produced prolonged hemodilution.

*Dextran.* In 1946 at the Presbyterian Hospital we were asked to study Swedish dextran. This material proved to be an effective means of increasing blood volume in both dog and man without changing the plasma electrolyte composition. However, we ran into a whole host of reactions with Swedish dextran. We had two severe antiphylactic reactions, one almost resulting in death. We stopped using the dextran and returned the grant back to the giver.

This year, in conjunction with Drs. Heidelberger and Kabat, we undertook the comparative study of native and Swedish dextran. (Table 1.) A group of medical students were skin tested and showed a reaction in 25 per cent. This confirms the work of Hehre and Neill of Cornell, and of others, who have proven that dextran is antigenic and that those who have had illnesses caused by pneumococcus 2, 12 or 20, or who have had typhoid, salmonella or certain streptococcal infections in the past, are not suitable subjects to receive dextran. In an emergency it would be impossible to screen these probable reactors, as no quick, reliable test has been perfected.

*PVP-macrose.* Figure 2 shows the electro-

* Supported by a grant to Columbia University by Schenley Laboratories, Inc., New York, N. Y.

phoretic pattern of polyvinylpyrrolidone in serum. PVP does not move in the electrical field and does not mix with plasma proteins. Ultracentrifugation of PVP gives a distinct pattern apart from plasma proteins, thus showing they are immiscible. Titration curve of polyvinylpyrrolidone reveals no acid

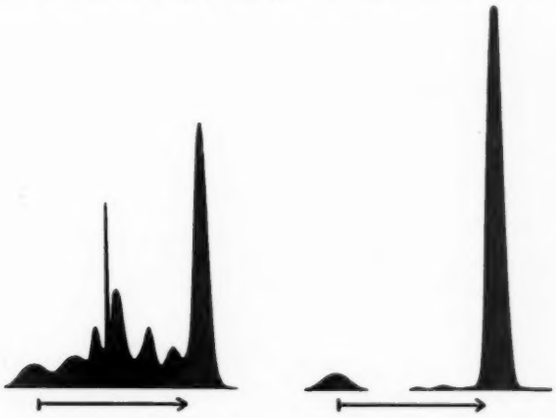



FIG. 1. Electrophoretic patterns.

or basophilic groups. In other words, it is an inert substance. Excretion data on a burn patient having received 2,000 cc. of 3.5 per cent of PVP in Ringer's solution reveal an output of 40 per cent in the first twenty-four hours.

PVP-macrose builds up plasma volume almost as effectively as albumin because each gram of PVP will bring into circulation 18½ gm. of water. By the end of twenty-four hours the blood volume has returned to normal. (Fig. 3.)

TABLE 1
SKIN SENSITIVITY TESTS

| | No. of Volunteers | Positive Wheals | Negative Wheals | Positive to Native Dextran |
|---|---|---|---|---|
| Native dextran | 51 | 25 | 26 | 25 |
| Swedish dextran | 26 | 5 | 21 | 5 |
| Pneumococcal polysaccharides (solutions containing 0.1 mg.%) II | 51 | 9 | 42 | 7 |
| Pneumococcal polysaccharides (solutions containing 0.1 mg.%) XII | 51 | 11 | 40 | 7 |
| Pneumococcal polysaccharides (solutions containing 0.1 mg.%) XX | 51 | 23 | 28 | 21 |
| Controls (phenol-saline) | 51 | 2 | 49 | 1 |

Following 1,000 cc. of PVP-macrose the plasma electrolytes concentration of potassium, chloride and $CO_2$ shows practically no change. (Fig. 4.)

In conclusion, clinically PVP-macrose is shown to be effective in the treatment of shock. (Fig. 5.)

Figure 6 shows the cases of four patients with traumatic and hemorrhagic shock treated at Harlem Hospital. To date we have treated seventy-eight patients with traumatic, hemorrhagic and operative shock effectively.

In treating the emergency case PVP-macrose

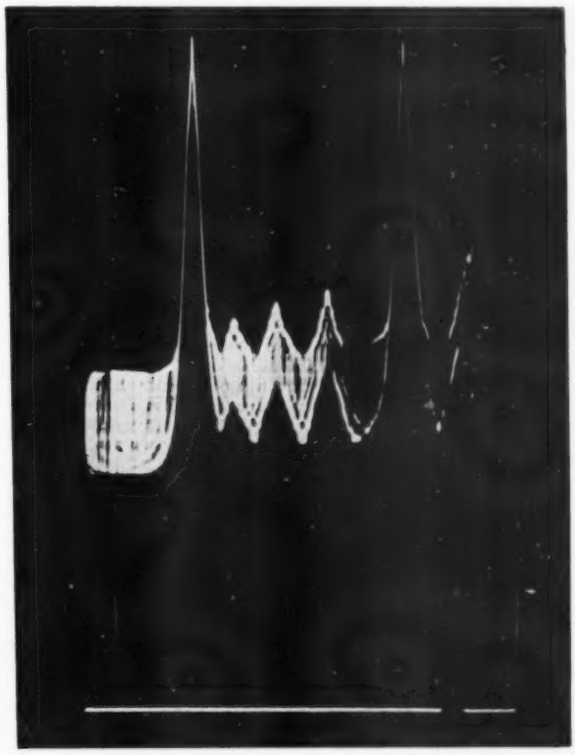


FIG. 2. Electrophoretic pattern of PVP and plasma.

gives the blood bank technician time to ascertain the proper blood group, correct Rh type and subtype, and to perform the adequate cross match by the indirect Coombs technic. In other words it also gives the blood bank time to secure the right type of blood. This eliminates the danger of an immediate kidney shutdown or of sensitizing the patient for all time.

PVP-macrose has proven to be effective clinically and we have it in use at our hospital.

SAM F. SEELEY (closing): I am sure everyone will agree that we are in a very poor state of preparation in case of national catastrophe, thinking primarily of the atom bomb. It is essential that greater subsidy be given to the extension of our information on the plasma expanders now available to us and to develop other plasma expanders free of some of the objections raised to the ones currently in use.

I would emphasize two points: First, we are so impressed with the high incidence of virus hepatitis after the administration of plasma that at Walter Reed Hospital we use it only in the most grave emergency. Currently, 300 to 500 individuals

may contribute their plasma to a single pool, thus increasing the chance of contamination by the virus which produces hepatitis.

Another fact is that one of the plasma expanders should be available in all operating and emergency rooms to take care of the time lag between the

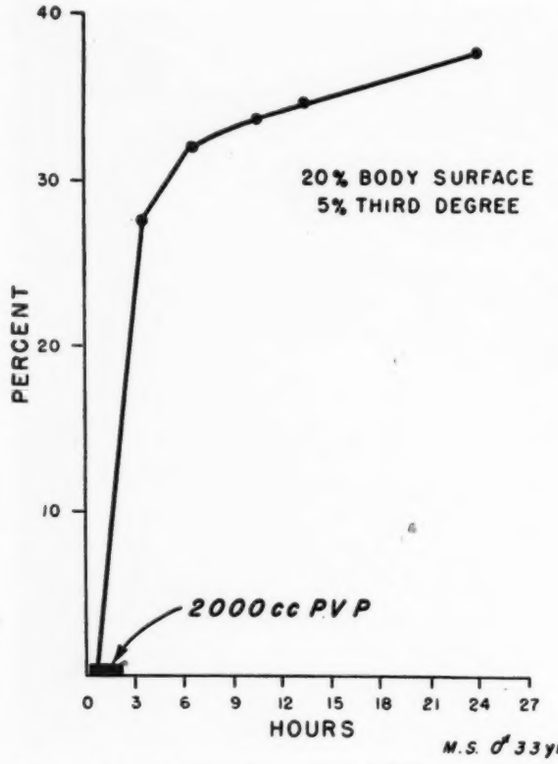



FIG. 3. Urinary excretion curve of PVP; severe burn case.

rooeipt of the patient in shock and the time that blood becomes available. It is also of importance to remember that the plasma expanders are effective for a good many hours in the absence of blood but that red cells will be demanded in proportion to the amount lost. Hemorrhage in excess of 40 per cent of total blood volume may demand the early administration of red cells.

The use of expanders and blood, however, may suffice to reduce the amount of whole blood required in multiple wounds. Many of our patients in Korea have received 20 to 30 pints of blood within the first twelve to twenty-four hours after very severe injury. When you exceed 5 to 10 pints of blood in transfusion, there is a multiplication of the opportunity of giving blood which is more than fourteen days old. Thus you give blood having a higher content of hemolysis which will administer to the patient hemolyzed blood in the form of hemoglobin and raise the potassium level, thereby increasing the chance of potassium intoxication.

Suffice it to say, the plasma expanders are doing the job with a great degree of safety where blood is not present.

Lastly, even saline solution, plasma or plasma expanders may be given by the arterial method in the patient who has no blood pressure or pulse. Thus it may be effective in raising the blood pressure to maintain the patient for the few moments until one of the better substances may be procured.

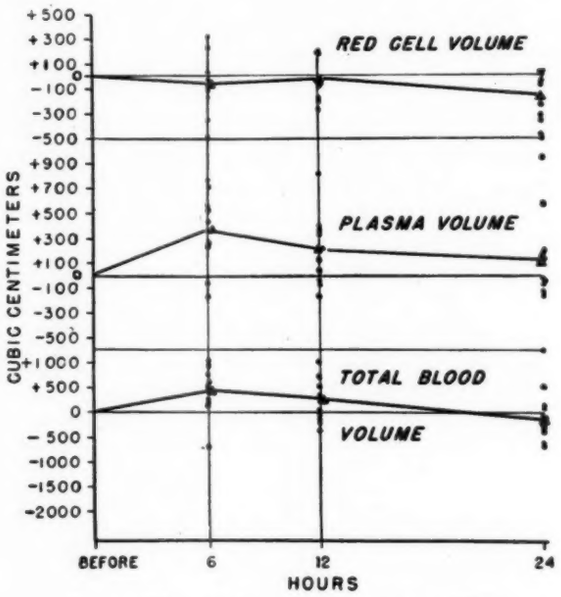



FIG. 4. Blood volume after 1,000 cc. PVP.

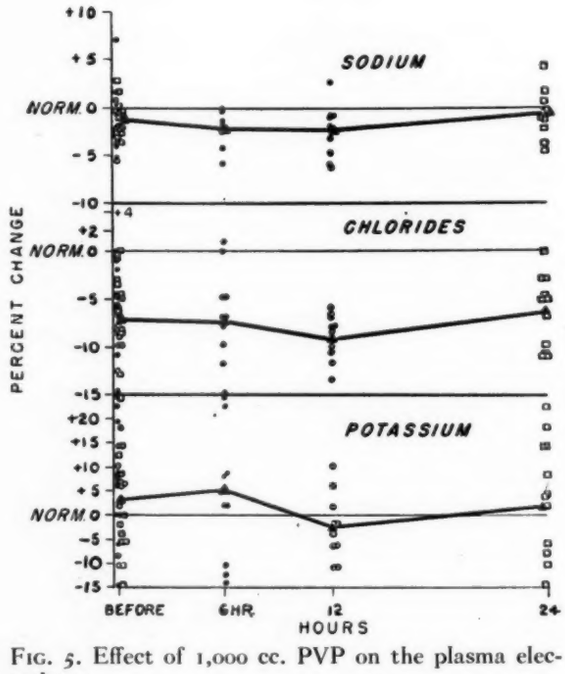



FIG. 5. Effect of 1,000 cc. PVP on the plasma electrolytes.

CHARLES L. FOX, JR. (New York, N. Y.): These two papers seem to complement one another so well I think it important to draw attention to their interrelation.

The first paper mentioned the important point that blood volume is reduced in shock. However, Dr. North called attention to something which unfortunately is all too frequently overlooked in trying to understand what happens in a severe injury. The point Dr. North made, confirmed by and the more detailed chemical studies have shown that there are disturbances beyond the vascular volume. Perhaps attention to some of these chemical disturbances outside the blood volume are worthy of further attention.

We should consider carefully the grand result

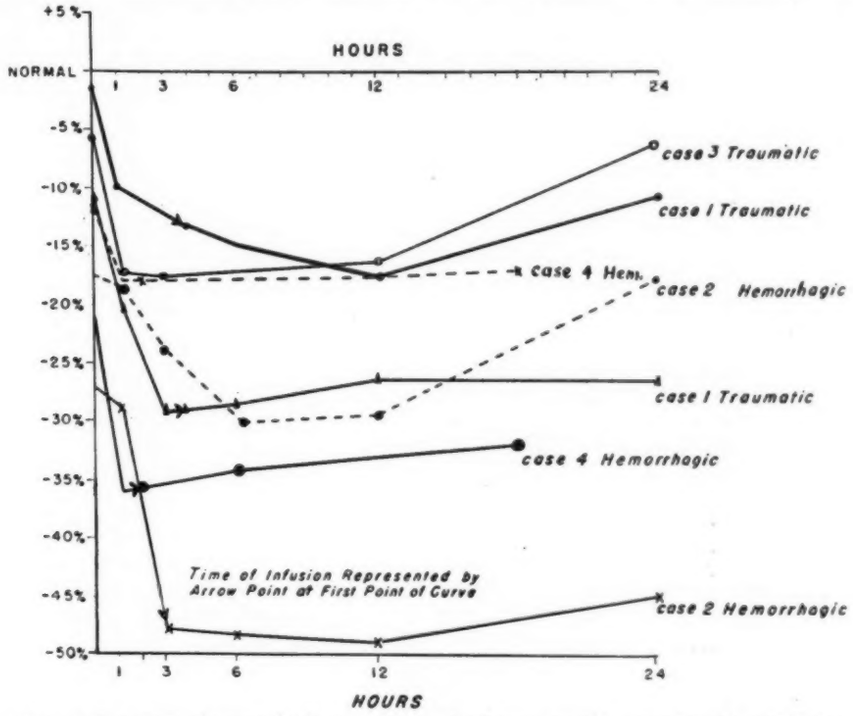



FIG. 6. Per cent changes in hematocrit and plasma specific gravity after infusion of 1,500 cc. PVP in shock.

his case, is that attention to the extracellular volume is also necessary. As a matter of fact, if one reviews the therapy which his patient received, it seems extremely interesting that the major therapeutic solution used in greatest volume was a balanced electrolyte solution approximating the composition of extracellular fluid.

Suppose Dr. North had followed what is tending to become the accepted practice of paying attention only to the vascular volume? Suppose his major therapeutic effort had been directed at expanding the blood volume by administering a solution which has as its chief function remaining in the vascular volume? One has to ask the question whether or not the result would have been so successful.

It becomes more and more obvious as further federal funds are used to investigate the problem of shock that there are enormous gaps in our knowledge as to the etiologic factors in shock. While we are temporarily justified in the rather simple and direct approach of trying to expand the vascular volume because all the data show that the blood volume is reduced, as physicians we must remember that the body mechanisms are infinitely complex and many things are not visible to the naked eye; that Dr. North achieved by paying attention to this extravascular volume and by using a large amount of a solution which has as its function the replacement of the sodium, chloride, bicarbonate, calcium and potassium, and perhaps the magnesium, which are important components of extracellular fluid.

WILLIAM H. AMSPACHER (Fort Sam Houston, Tex.): Dr. North certainly must be congratulated on being such an astute clinician as to recognize and determine what actually happened to this patient. Unfortunately, everyone who treats a great number of patients with major trauma is confronted by similar situations every year. The proper diagnosis is not being made for many of these patients and they proceed to a fatal termination, are signed out as cases of unremitting or irreversible shock when actually they are dying of some unrecognized condition which could have been corrected.

The greatest advance in history for the treatment of traumatic shock was the establishment of the blood bank. However, even the use of whole blood has its shortcomings. Instead of performing a careful physical examination and attempting to discover all the physiologic and anatomic altera-

tions, many physicians have been guilty of depending almost completely on blood to cure their patients. I predict that Dr. North's paper will inaugurate a series of similar case presentations.

In Colonel Pulaski's paper I believe that too much emphasis has been placed on the characteristics of an "*ideal*" plasma expander. The fact that we will not have an adequate supply of whole blood if we are ever confronted with a real major catastrophe must be recognized. An adequate supply of materials capable of restoring blood volume must be available if we are to cope with conditions encountered in wound shock. An attempt should be made to improve the plasma expanders but we must not hinder production or stockpiling while awaiting the "ideal" any more than we must abandon the use of whole blood because an occasional fatal transfusion reaction is seen.

Recently Dr. Anthony R. Curreri, Lieut. Thomas M. Cloud and I took a large supply of dextran (expandex, Commercial Solvents Corporation) into the field in Korea for use in the wounded. Dextran was used in sixty-one cases; the largest amount administered to any individual was 4,500 cc. in a period of thirty-six hours.

Recovery occurred in patients in whom hemodilution lowered the hematocrit as low as 11 per cent. Whole blood was given in addition to expandex when such severe hemodilution was present.

There has been some concern about the effect of dextran on the kidneys; and although careful investigators have been able to show a transient minor interference with kidney function, this has not been manifest clinically.

The urinary output was very carefully determined for all the casualties in Korea treated with expandex. Each patient had a urinary output in excess of 1,000 cc. per day. The condition of many of the casualties who were severely wounded was typical of those cases in which a urinary shutdown is always feared.

I believe that the mild diuretic effect of a plasma expander may not be harmful, as the diuresis probably results from the excretion of free water which has been made available as the result of a larger blood volume and better circulation through the kidneys. The plasma expanders can be used freely and the water administered as a result thereof is probably beneficial. In the treatment of shock there is a tendency to give too little water in addition to blood.

Fraser N. Gurd (Montreal, Canada): On behalf of those of us from Canada I would like to thank Colonel Pulaski for this opportunity to be brought up to date on the question of plasma expanders. We are very grateful.

I would like to commend Doctor North on his remarkable case. There is certainly a place for recognition of a "third space" effect in visceral injuries which result in a large output of fluid into the peritoneal cavity. Although the extracellular space must be attended to just as much as the circulating intravascular volume, I believe it may be unwise to attempt complete refilling of a badly depleted extracellular space in a short period of time such as five or six hours. If one presses too hard, patients will die from wet lungs and heart failure. So far as fluid and electrolyte are concerned, aberrations on the low side are better tolerated than overloading.

As in the case mentioned, one is sometimes in the position of having given all the blood, fluid and electrolyte which should be necessary (that is to say, all spaces may be considered to have been properly filled); yet because of the underlying exhaustion of the patient extensive surgery results in relapse of the blood pressure. In such circumstances I believe there is a place for a continuous drip of nor-epinephrine, which we have found effective in sustaining blood pressure until the compensating mechanisms have had time to stabilize.

Edwin J. Pulaski (closing): As regards dextran, permit me to reiterate that our experience with the foreign-manufactured material was the same as Dr. Scudder's. On the contrary, the overall experience with American clinical dextran is that the incidence of untoward reactions is small and it is an effective plasma expander, as Colonel Amspacher reported with his Korean and stateside experiences. I now have it on good authority that the Swedish manufacturers have gone to the American type of organism used in the synthesis of dextran and to American methods of refinement of the product for clinical use.

Certainly everyone will agree with Dr. Fox that the use of electrolytes is important in the adjunctive therapy of shock. I would like to remind you that the different plasma expander solutions contain electrolytes. The specifications for the maximum amounts of these electrolytes in a unit of solution are: potassium, 25 mg. per 100 cc.; magnesium, 4; calcium, 12; inorganic phosphorus, 8; and sodium, 360.

John Paul North (closing): I must disclaim any credit for the unerring discernment that Dr. Amspacher attributed to me and also point out, as I tried to do in the paper, that we made a number of errors in management as we went along. I did not indicate the reason for our reluctance to use more blood in the early stage although we were urged to do so. Only a month before, in handling a quite similar case of perforation of the duodenum, we attempted to do so mainly by the use of blood transfusion and we were all thoroughly convinced that we had killed the patient by over transfusion. In this case we had reason for swinging to the other side and it turned out more happily.

# ILIAC BONE GRAFTS REPLACING TANTALUM PLATES FOR GUNSHOT WOUNDS OF THE SKULL*

CLIFFORD L. KIEHN, M.D. AND ANTONIO GRINO, M.D.
*Cleveland, Ohio*

SUBSTITUTES for living tissue transplants have been used in various recipient sites to correct traumatic deformities for many years. This is particularly true for the restoration of skull defects acquired during military service and after each war a new foreign material is used for their correction.

During a period before and after World War I silver and celluloid were used extensively with varying degrees of success. Erdman in 1903[1] first used celluloid which was prepared with no nitric acid and urea as this allowed the material more elasticity for correction of midline frontal bone defects. This substance was used as late as 1937 by Pringle,[2] who pointed out that its great disadvantage was the foreign body reaction of the host. Noon[3] in 1917 used silver plates in three cases of skull defects with good results.

During the interval between the two wars bone and cartilage were used quite successfully. Nesselrode[4] in 1919 and later many others used tibia grafts to the skull and Gallie[5] stressed the use of fresh living bone grafts with the addition of bone chips around the edges of the main graft.

During and after World War II acrylic resin[6] but more extensively Tantalum was used for correction of these deformities. More recently pure polyethylene is being employed because it is more inert and can be molded in hot water to fit irregular defects. Several cases have been reported by Alexander,[7] but these plates have not been in place long enough to assess their value properly.

In two years following World War II Levin[8] placed 128 Tantalum plates in skulls for various deformities. Ninety-four of these were satisfactory, eleven of which were removed for infection or cosmetic failures with no increase or diminution in the incidence or severity of traumic epilepsy. The incidence of infection in the entire series was 7 per cent.

Several reports have been published for failure of Tantalum plates used to correct deformities of the frontal bone which included the supraorbital ridge. Work[9] in 1949 reported eight cases in which complications arose when the metallic plate was placed in this region without unablated frontal sinuses.

Lane and Webster 1947[10] collected data for the Veterans Administration where 115 plates were inserted. Fifty-two plates were removed and of these twenty-six, or 50 per cent, had been inserted in the frontal region and eighteen of these, or 35 per cent, involved the frontal sinus.

We have found similar results in which Tantalum plates were removed for the following reasons: (1) Infection, which probably ascended from the nasal cavity through the frontal nasal duct causing abscesses around the plate; (2) deleterious effects of extreme heat and cold causing pain to the area underlying the plates; (3) pain and headaches; (4) erosion through pedicle flaps and into the orbit; (5) cosmetic results in which an attempt was made to correct a facial deformity in continuity with the skull defect.

The deformities produced by removal of the plates leaves the brain unprotected and a cosmetic disfiguration that calls for a type of material that is easily obtainable and one that will maintain viability for their correction.

We believe that the iliac bone is an excellent donor site and fulfills those requirements. It is large enough to correct defects equal to half the skull and its shape lends itself very well for the restoration of the frontal region. The crest of the ilium serves as an excellent supraorbital ridge and the ala can be used for extensive defects of the frontal and parietal regions. It is not usually necessary to utilize the full thickness of the ala thereby exposing a larger cancellous surface to an area from which it will

* From the Dept. of Surgery, Plastic Section, School of Medicine of Western Reserve University, and Crile Veterans Hospital, Cleveland, O. Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the results of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

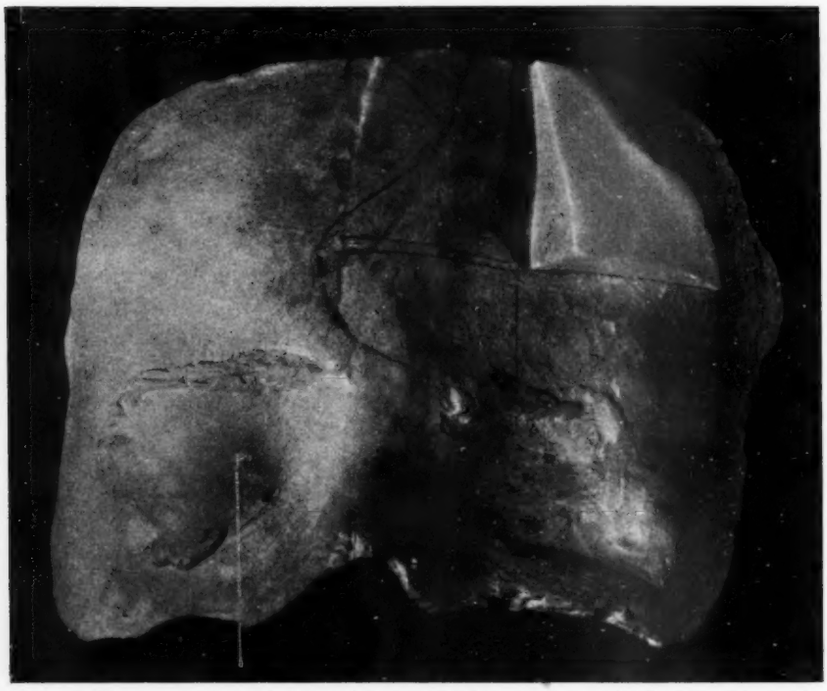


FIG. 1. Plaster cast of defect with sterilizable pattern which determines the size and shape graft needed.

become more rapidly vascularized. This assists in the maintenance of viability. The defect remaining after removal of the graft leaves very little disability and regenerates new bone to some extent.

Several principles must be followed for successful results if fresh autogenous bone grafts are used. If the plate has been removed because of infection, obviously no attempt must be made to correct the defect until all signs of the infection have subsided and the frontal sinuses ablated to prevent potential infections from the nose if there is communication between the two.

The bone graft should not be placed under scar tissue which will prevent early vascularization of the graft and also provide an area through which it might erode. The cicatrix should be excised and closed with normal tissue by means of rotation flaps or if the area is too large a pedicle flap must be brought in from another source.

## TECHNIC

The restoration of the contour in the frontal region is usually done with the patient receiving a general anesthetic through the endotracheal route. If two operating teams are available, one will take the graft by making an adequate incision over the crest of the ilium and by blunt dissection, the muscles and periosteum are stripped from the bone. The size and shape of the graft can be predetermined by constructing a plaster cast of the defect before the operation. (Fig. 1.) Lead plates are shaped to equal the defect in two diameters, sterilized and used as patterns during the removal of the graft.

The preparation of the recipient site is done through a coronal incision which allows adequate exposure for comparison with the normal side while shaping the graft and also prevents a disfiguring vertical scar on the forehead.

As the scalp flaps are turned back and the defect approached, care should be taken not to expose the frontal lobe as it is covered with a false membrane (Fig. 2) which has encapsulated the Tantalum plate. This is left attached to the brain but dissected away from the edge of the defect so that the graft will come in contact with the skull.

The periphery is then freshened with an osteotome by producing a ledge for the graft to rest upon. Drill holes are then placed in strategic positions in the skull and graft. The transplant is then shaped and held in place with No. 28 stainless steel wire. If there are any irregularities around the edge, these are

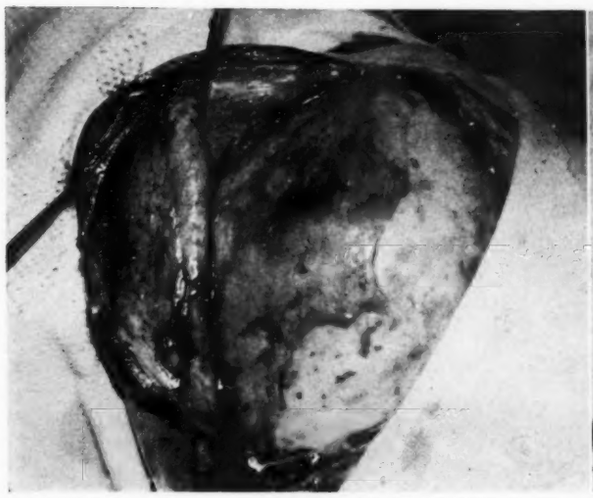


FIG. 2. Membrane over frontal lobe which encapsulated tantalum plate.

FIG. 3. Bone graft and chips filling skull defect.

FIG. 4. Tantalum plate eroding into right orbit; pedicle graft in forehead covering plate.

FIG. 5. Postoperative bone graft to frontal and supraorbital ridge replacing tantalum plate.

filled with cancellous bone chips. (Fig. 3.) The scalp is then closed.

The postoperative complications have been negligible in the ten cases we have done. A small hematoma developed in one patient and a bone chip extruded from the edge of the graft in another patient. They have all been relieved from the symptoms for which the plates were removed.

## CASE REPORTS

CASE I. C. E. was injured in 1945 when hit by a machine gun bullet which destroyed the right frontal soft tissues and bone. That same year he had a tube pedicle and a Tantalum plate placed in the defect. He was symptom-free until August, 1948, when headaches and an infection in his right eye developed. In February, 1949, the Tantalum plate was removed because it could be seen extruding through the (Fig. 4) conjunctiva under the upper eyelid. Sight in that eye was normal. X-rays (Fig. 6) revealed a large Tantalum plate in the frontal region with no evidence of osteomyelitis. In May, 1949, an autogenous iliac bone graft was placed in the defect which healed without complication. (Fig. 7.) The frontal sinuses were ablated preventing infection ascending from the nose. The following year the entropion caused by the scar formed around the plate was corrected with an oral mucous membrane graft. (Fig. 5.) The eyebrow can be brought down to a normal position with a "Z" plasty.

CASE II. R. S. sustained a shrapnel wound in the left eye and frontal region in January, 1945, which was débrided and closed. In May, 1945, a Tantalum plate was inserted in the frontal defect. One year later the patient began to have recurrent headaches with changes in the weather, particularly from extreme heat and cold. He was then seen by us because of headaches and tenderness around the plate. (Figs. 8, 11.) In May, 1952, the plate was removed (Fig. 9), and an autogenous iliac bone graft placed in the defect two months later. (Fig. 12.) Healing was uneventful except for a small hematoma and there has been complete cessation of headaches. (Fig. 10.)

CASE III. W. M. was struck by a mortar shell

FIG. 6. Tantalum plate in frontal region.

FIG. 7. Iliac bone graft replacing tantalum plate.

8

9

10

FIG. 8. Tantalum plate bulging in forehead which was very tender to palpation.

FIG. 9. Tantalum plate removed because of headaches and pain brought on by hot or cold weather.

FIG. 10. Iliac bone graft to frontal region, restoring contour.

in 1945 which resulted in the loss of the left eye, periorbital, frontal and temporal bone. He was admitted in 1949 for therapy of epilepsy and correction of the orbital defect. Epilepsy and headaches developed after insertion of a Tantalum plate in 1946 and the patient was placed on dilantin. (Fig. 13.) In February, 1950, the Tantalum plate was removed because of improper contour, headaches and epilepsy. (Fig. 14, 16.) An autogenous iliac bone graft was done to protect the frontal and temporal lobes of the brain. To improve the contour a tube pedicle flap was brought up from the abdomen so that the second bone graft to the region would have adequate soft tissue coverage. (Fig. 17.) X-ray (Fig. 15) shows the bone graft in place. A small sequestra was removed after the original graft followed by complete healing. During the last bone graft the frontal sinus was

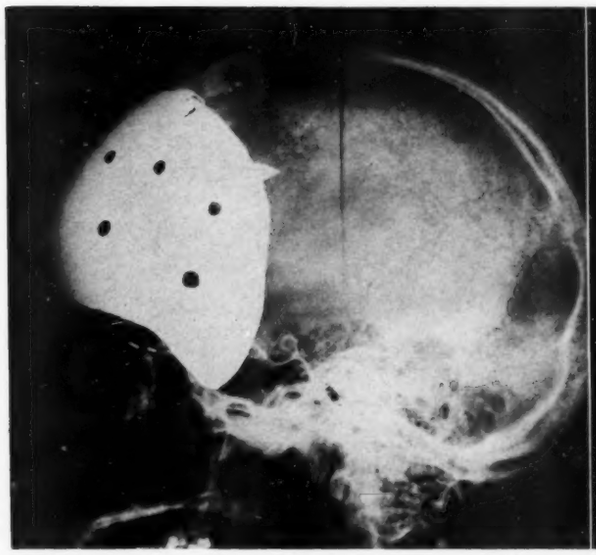


FIG. 11. X-ray of tantalum plate in frontal bone.

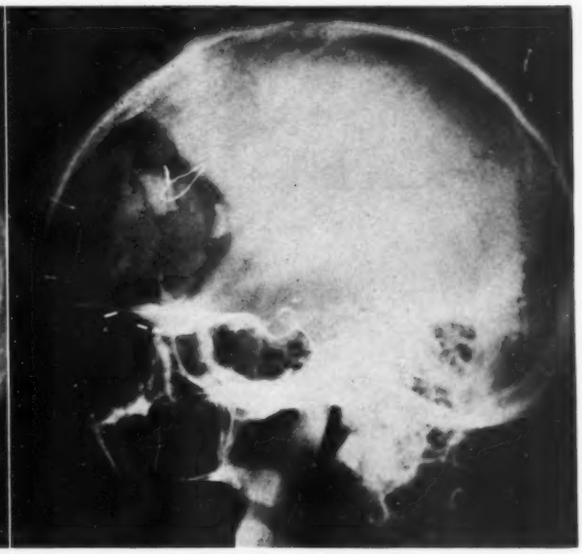


FIG. 12. X-ray of bone graft to frontal defect.

13 14 15

FIG. 13. Tantalum plate in frontal and temporal region.

FIG. 14. Defect after removal of tantalum plate for headache and epilepsy.

FIG. 15. Bone graft and tube pedicle which will require further adjustment.

inadvertently entered and will probably have to be ablated as up to that time it was not involved in the defect. The patient now wears a conformer in the left orbit and further plastic procedures are being carried out to restore contour. He discontinued anti-convulsive medications in March, 1950, and has not had any convulsions or headaches since that time.

## COMMENTS

The principle of replacing lost structures with autogenous tissue, if available, for the restitution of function without the complicating factor of foreign body reaction should be closely followed. The organism has an antipathy to any foreign body and Tantalum is no exception. This is particularly true when placed in the frontal region because of the possible added danger of infection from the sinus and, as this is a comparatively kinetic area, the motion will cause erosion through the tissues in many instances.

Autogenous iliac bone grafts become an integral part of the host and probably remain viable from the time of transplantation[11] to complete healing. The source of bone for the autogeneous transplant to the skull is adequate for the largest graft as bone may be removed bilaterally without disabling the patient. The bone is no more difficult to shape than Tantalum and there is a greatly reduced risk of complications.

## SUMMARY

1. Tantalum is a foreign body and with the added danger of infection from the sinuses it

16 17

FIG. 16. X-ray of tantalum plate in frontal region.

FIG. 17. X-ray of iliac bone graft to frontal and temporal region.

should not be used in the frontal region of the skull.

2. All patients following removal of the Tantalum plates were relieved of their symptoms.

3. There was no recurrence of symptoms when the deformities were corrected with autogenous iliac bone grafts.

4. Iliac bone is adequate for the protection of the frontal lobe and restoration of contours.

5. With an adequate source of autogenous bone it is not necessary to use an alloplastic transplant, which, with slight trauma, may become infected, absorbed or extruded.

REFERENCES


1. ERDMAN, J. F. Gunshot wounds of the skull. *Ann. Surg.*, 37: 607, 1903.
2. PRINGLE, J. H. Treatment of acquired defects of the skull. *Brit. M. J.*, 2: 1105, 1937.
3. NOON, C. Repair of large bony defects in the skull by means of a metal plate. *Lancet*, 1: 373, 1917.
4. NESSELRODE, C. Closure of cranial defects by osteoperiosteal grafts taken from the tibia. *S. Clin.*, 3: 789, 1919.
5. GALLIE, E. and ROBERTSON, D. E. The transplantation of bone. *J. A. M. A.*, 70: 1134, 1918.
6. ELKINS, C. Cranioplasty with acrylic plates. *J. Neurosurg.*, 3: 1999, 1946.
7. ALEXANDER, E. and DILLARD, P. The use of pure polyethelene plate for cranioplasty. *J. Neurosurg.*, 7: 492, 1950.
8. LEVIN, W., GRAHAM, M. P. and NORTHCROFT, G. B. Tantalum in the repair of traumatic skull defects. *Brit. J. Surg.*, 36: 26, 1948.
9. WORK, W. P. Paranasal sinus in relation to skull injury. *J. A. M. A.*, 141: 977, 1949.
10. LANE, S. and WEBSTER, J. E. Early results in tantalum cranioplasty. *J. Neurosurg.*, 4: 526, 1947.
11. KIEHN, C. L., et al. A study of the vascularization of experimental bone grafts by means of radioactive phosphorus and the transparent chamber. *Ann. Surg.*, 136: 404, 1952.

# BONE GRAFTS FOR LARGE GAPS IN MANDIBLE*

JAMES BARRETT BROWN, M.D. AND MINOT P. FRYER, M.D.
*St. Louis, Missouri*

IN gunshot wounds and other injuries in which there are large losses of substance about the face and mandible, there are the essential problems of reconstruction of the soft parts and of bridging the gap in the jaw with a bone graft.[1] The objects of surgical rehabilitation are a watertight mouth, a chance of function of the jaws, restoration of speech and trying to produce the most normal-looking contour anatomy. The term "contour anatomy" is one we have used to describe an important function of the face, that of looking natural enough to permit a normal educational, social, economic and marital life. This might be said to be the fourth function of the face, the other major ones being eyelids to protect the eyes, a nose with a normal airway, and the mouth and jaws to permit eating. This function of normal contour anatomy is of great importance in gunshot wounds in young people in war services as they have given so much of themselves and have so far to go in life.

In rebuilding the lower third of the face, including the bone and soft parts, the process has to be in reverse order of usual plans of mechanical construction. In a building the support is put in first and then the walls added, whereas in plastic surgery reconstruction the covering has to be put in first and the support or armature put in underneath it, possibly comparing it to a tent rather than a solid building construction. In other words, although the bone graft may eventually hold the soft parts where they belong, the soft parts must be present in adequate amount and supply adequate minute blood supply for the bone graft to take and persist.

Such a broad policy or philosophy of approach to the problem is necessary or concepts of repair may not result in successful outcomes. The supplying of an adequate minute blood supply free of excess scar tissue is a basic requirement. In a previous paper[2] a young soldier was illustrated who had had four grafts done for a mandibular loss before a permanent one was obtained. This was finally obtained by resection of the whole scarred area and substituting a permanent pedicle blood-carrying flap in the area brought up from lower down on the neck. This gave a suitable bed of soft tissue for the graft to reside in and an adequate source of minute blood supply for nourishment.[3] Bone grafts cannot be routinely successful with insufficient covering or scant blood supply and it is again seen to be true that "the deep healing can be no better than the surface healing." This same finding has been observed and reported in lesions of the extremities requiring bone grafts and tendon repairs.[4,5]

In these extensive facial repairs that finally mean so much in the patient's life there are a great many important considerations. They can be summarized as follows and the description of a typical repair can be outlined, not as a single case report, but as illustrative of plans and procedures for surgical rehabilitation of these unfortunate patients.

*Persistence* of the bone graft is dependent on a suitable bed of soft tissue and an adequate minute blood supply, and also on a completely healed wound without infection or tracts leading into the mouth or to the outside surface. In other words there must be a clean, healed, soft area that again can heal after the bone graft has been inserted.

*Bridging a gap in a bone* is a much more extensive and difficult procedure than holding two un-united fragments together until healing has occurred, and the bridge of bone in large gaps of the mandible is what is necessary if the remaining fragments are to be aligned and any degree of function and normal appearance is to be obtained.

*Permanent persistence of these large long bone grafts*, as such, is thought to be the situation in these bridges of bone. Although they may gradually be replaced in metabolic processes, they do not seem to be absorbed or replaced

* From the Department of Surgery, Plastic and Maxillo-Facial Surgery Service, Washington University School of Medicine, St. Louis, Mo.

with bone from the ends of fragments. The wire fixation and the size and shape of these long bone grafts are retained after many years on x-ray examination and it is probable that they are true grafts, persisting as skin grafts do. (Fig. 3.)

*Preparation* and support *of fragments* of the jaw and of teeth for grafting is important and dental assistance may be needed. The graft should bridge a gap of the original size of the loss instead of just going across collapsed fragments, and in obtaining and holding position of the two ends dental splints may be of service. If no teeth are available, this dental assistance cannot be counted on; and so the preservation of all possible teeth is another important dental problem.

*Dental prostheses* for later use also can give aid if teeth can be saved and the usual "turn in" of both fragments and graft can be prevented to some extent. The same dental splint that may hold fragments in alignment during the preparation period may be retained for help with fixation of the graft.

*Direct bone grafting and immediate permanent fixation of the bone graft to the fragments* may be carried out when there are inadequate teeth for interdental wiring support or for the retention of a dental splint.

*The graft becomes its own splint* in this instance and is possible by separation and alignment of the fragments into normal position and immediate direct wiring, in place, of the solid bone graft. Stainless steel wires usually suffice and persist without trouble but may require removal later.

Internal wires or onlay plates may be used to assist in this graft fixation. The internal wires may go through the graft, alongside of it or from side to side in the remaining fragments.

External splints have been reported by others as useful in bone grafting, but the more direct method of fixation and having the graft act as its own splint has been more applicable in our hands.

Artificial subcutaneous prosthesis of metal or plastic, chip bone grafts with or without metal trays, foreign bone grafts, fresh homografts, preserved animal or grafts from human bone banks have not been observed in an adequate enough series to be relied on routinely, or are not thought certain enough to rely on as substitution for the solid, fresh autograft for these large permanent bridges at present.

*Internal wire pins* or *steel bars* from fragment to fragment for support of a gap were first used in the immediate repair of a wide bone loss and may be used at the time of original treatment or in jaw resections for tumor. However, the final repair, especially in young patients, is thought to be best with a fresh autogenous bone graft that he can call his own and depend on.

*Source of grafts* is usually fresh autogenous ones from the ilium or rib; both of these supply cancellus bone that would seem to graft and persist better than a solid, dense, cortical graft from the tibia. Completely cortical (or chip) grafts to try to obtain a better blood supply for healing are thought advisable by some. However, they do not lend themselves to immediate solid fixation without tooth wiring or splint support, and the more solid type seems superior if the bed is adequate. Cancellous-to-cancellous bone might give the quickest union but it would still not allow firm, independent fixation unless some firm cortex were left for wiring. This is a technical point, however, that is decided in individual cases by different surgeons.

*Bone and cartilage* grafts are sometimes used when the whole side of the jaw is out, including the condyle. In this instance a section of the sixth or seventh rib is removed along with the attached cartilage. The angle is fairly normal one for the jaw contour and the cartilage end floats free in the glenoid region. There is no special joint fulcrum but the extent of fixation may be adequate for improved function and position.

Size of the graft in length does not seem to make much difference as long as a suitable one can be obtained and fixation maintained in a satisfactory bed. Single rib grafts from one ramus clear around to the opposite ramus have been successful. Bridging the whole side of the body is frequently done, with good stability and strength resulting. Very large grafts may occasionally be put in first and fixed to the fragments secondarily.

Immobilization of graft is from eight to twelve weeks according to needs. Function is often close to normal depending, of course, on the available teeth, the lack of severe "turn in" of the fragments and the success of the dental prosthetic appliance. These later may not find a good bearing surface in extensive injury and repair but the ingenuity of the

dental prosthodontist usually produces a worth while appliance.

*Final building out of the jaw* to restore normal profile may be done with onlay bone graft, cartilage or foreign subcutaneous prosthesis as desired.

*Regeneration of the jaw* has been noted and reported by us previously. It has been found that in infections there may be complete loss followed by complete regeneration of the body of the jaw. This, however, has not been seen to occur in traumatic loss or surgical removal and therefore is not relied on. In infections the reason is possibly because destruction and regeneration presumably are going on at the same time, whereas in trauma the whole area is lost at one time permanently, whether some periosteum has been left or not.

*The preliminary soft tissue preparation* and replacement is done before the bone graft is put in but is described here secondly because bone grafting has been taken as the main subject.

*Flaps* for repair of the soft tissues are designed according to the extent of loss and of scarring. Local flaps, as in all plastic surgery, can be considered but are not used for very large losses as they might result in further scarring and collapse of the fragments of the remaining jaw. Rather than this, it is better to fill the defect with soft tissue from a distance and let the bone fragments separate to their original normal positions.

A permanent pedicle blood-carrying flap can be brought up from lower in the neck and the pedicles left intact so there is a new blood supply to the area brought in permanently by the flap. This flap is used if there is not a very wide loss of tissue. The usual flap is somewhat of a drain on the blood supply of the area when the pedicles have to be divided.

Distant delayed pedicle flaps are brought up from the chest when there is a large loss. They have to be lined if there is not enough mucosa available, and the lining is turned in as a secondary flap or supplied as a thick-split graft under the pedicle when it is first raised. Flat flaps are usually relied on but the pedicles can be tubed if this will make shifting easier.

*Tongue fixation* out on the neck should be freed and mobilized before the flap or bone graft is done or the tongue may never be gotten free. Usually the scar can be resected and the tongue loosened to go back into place. The defect can be covered with a free graft, preferably a full-thickness one from above the clavicle to give the best function and softness, or with small local flaps. If there is extensive loss of tongue, there is all the more reason to try to get it as free as possible, as speech with a fixed stump may be very difficult.

*Salivary flow* out onto the chest has to be stopped before the bone grafting; and if there is no wide loss, the first consideration is to mobilize lateral flaps and close the floor so the saliva will go back down the throat instead of out on the chest. This is important as a morale builder because the patient who is freed from this constant annoyance believes that he has made great progress toward recovery. Just to get this flow reversed, however, is no reason for further distortion and collapse of the floor rather than adding a sufficiently large flap with necessary lining.

Excision of scars and closure in stages has almost no place in this type of injury. It collapses the fragments, fixes the tongue and may leave the saliva pouring out. A good deal of difficulty has been encountered (mouths being pulled open and saliva pouring out, eyelids pulled down from the globe and emotional expression interferred with) in wounds of the face when the principle of excision and closure has been applied to wounds of too great extent.

*Anesthesia* for this work is most important and needs careful consideration and cooperation. When safe general anesthesia is available, there is little use for local; but if necessary the whole work can be done under deep block of the fifth and cervical nerves and infiltration.

*Tracheotomy* may be already present and can be left open until the major work is done. It, however, may interfere with the work and, if so, the opening may be allowed to heal as soon as it is safe to do so.

*Pressure fixation* dressings are necessary throughout all these procedures as much so as almost any step in the procedure, both for the preliminary flap and for the bone graft.

*Joint and coronoid* fixation of the remaining jaw fragments is rare; but if it exists steps to relieve the fixation should be carried out first for there is not much reason to graft into an ankylosed jaw.[6]

*A fascial sling* put through an inert flap and anchored in each temporal region may be necessary to hold up the thick lip. So far no one has ever built a sphincter, which the mouth is, but substitution can be made for it. The larger

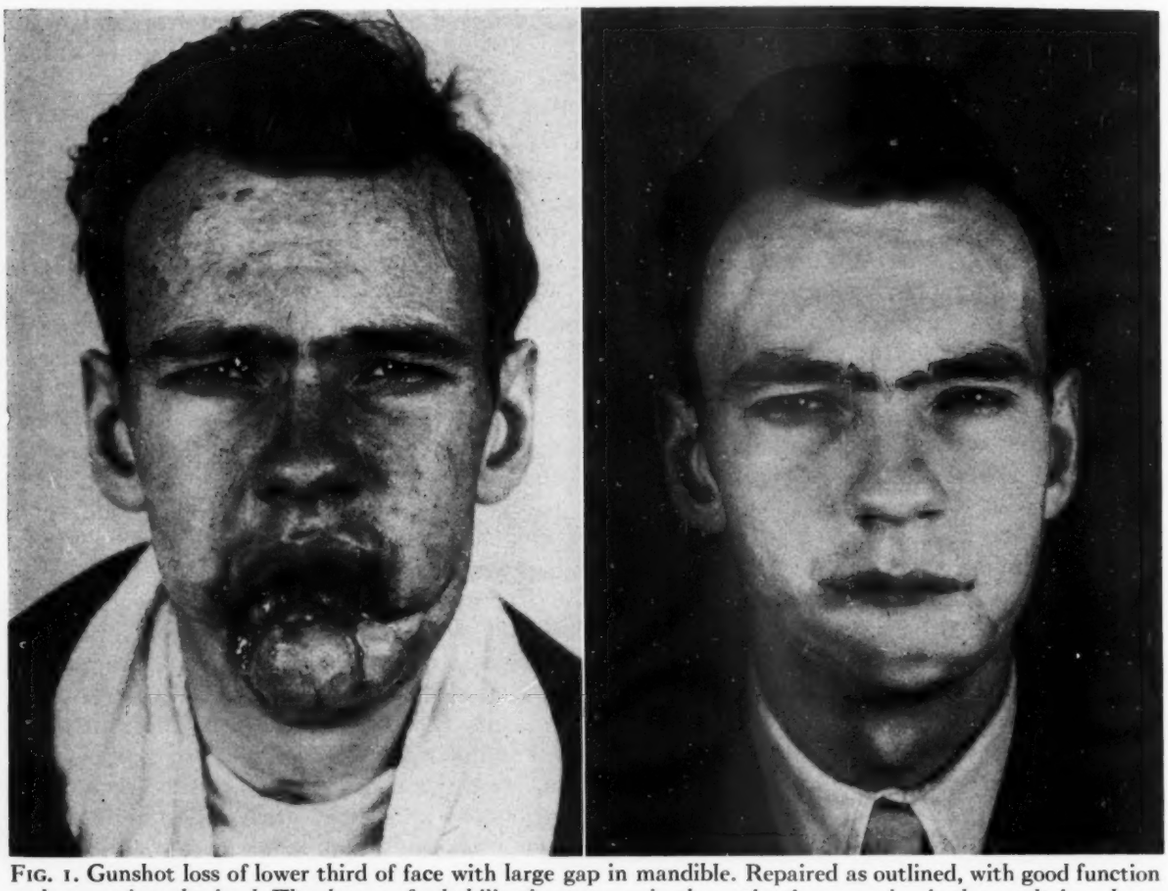


FIG. 1. Gunshot loss of lower third of face with large gap in mandible. Repaired as outlined, with good function and expression obtained. The degree of rehabilitation as seen in the patient's expression is the result in a large prrt, of the patient's determination.

flaps are attached high so the middle third muscles can help, and there has to be a balance between some limitation of opening and the mouth's remaining watertight, and the mouth's being watertight but still able to be opened.

Tattooing or pigment injection may be useful to assimilate a vermilion, and *speech training* may be required if there is difficulty in the patient's use of what is left for him to work with.

These patients fortunately suffer but little pain at the moment of the accident even though the fifth nerve roots are torn out. However, there is agony and despair to follow, and the challenge of surgical restoration and of their own rehabilitation is one of the most demanding in surgery of this type.

Final good surgical restoration is usually possible, and inspiring rehabilitation, mainly by the patient's own effort, is seen in most of these unfortunate patients.

The case illustrated is described in full, not as a case report but as a typical example of war injury and repair. (Figs. 1, 2, and 4.) In this boy there had been early saving of life in the first echelon by forward surgical officers and nurses and men, and with immediate tracheotomy. Late surgical restoration included a delayed flat flap from across the chest, with a side flap turned in from above to line the flap and form the lining of the inside of the mouth. Because of wide loss and not wishing to collapse the bone fragments further, the salivary flow onto the chest was not reversed until the flap itself was put in place to make the mouth watertight. (Figs. 1, 2 and 4.)

After the flap was solidly in place, soft and without induration, a large (12 cm.) bone graft from the rib was put directly in place, utilizing its own solidity and solid wire fixation for splinting so that the graft became its own splint. In this instance there were no teeth for fixation but no internal wires or external splint was necessary, just the graft itself wired in solidly

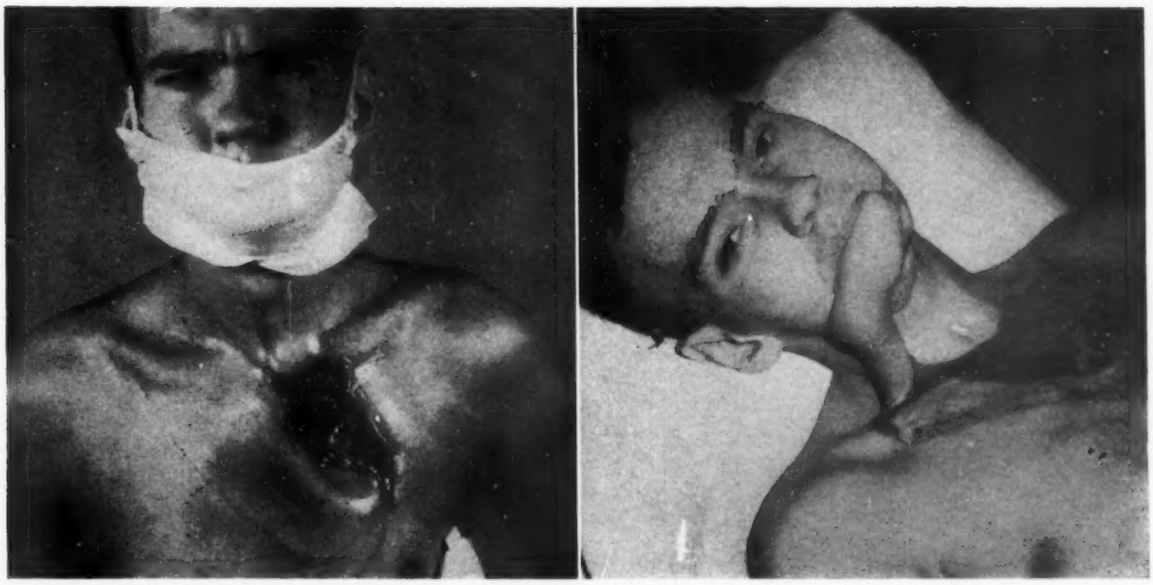


FIG. 2. Flat flap delayed on chest in three stages with side flap turned in from above for lining the mouth.

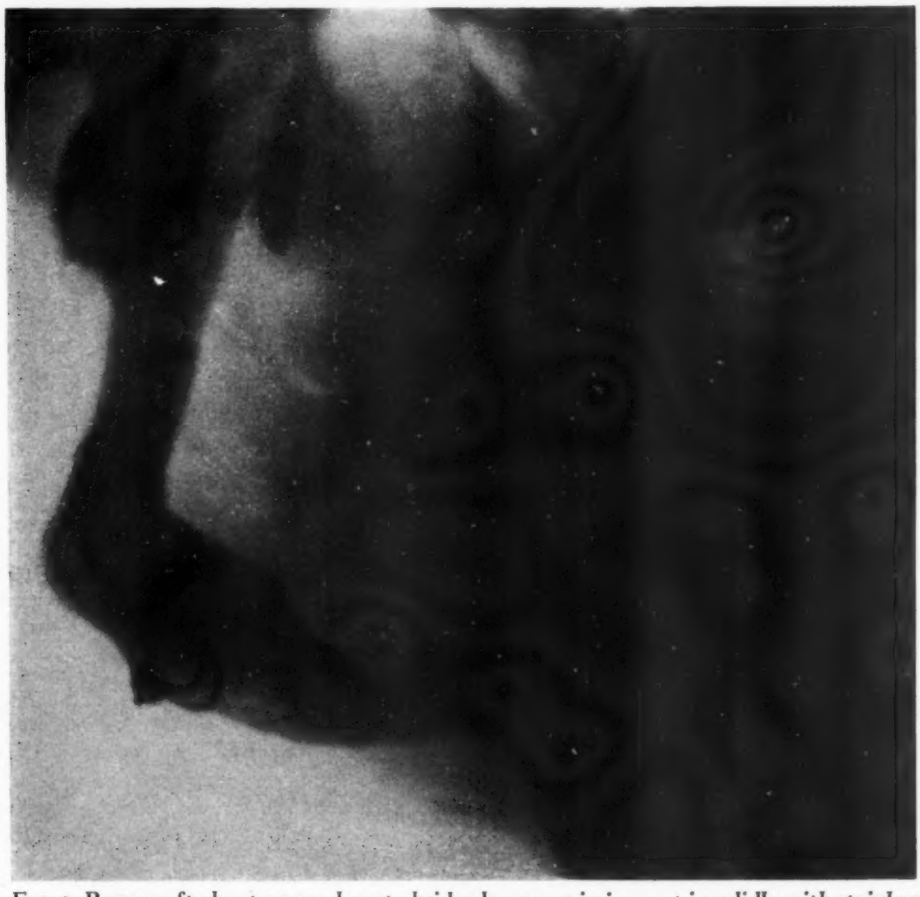


FIG. 3. Bone graft about 15 cm. long to bridge large gap in jaw put in solidly with stainless steel wire. The graft acts as its own splint, only other fixation being a pressure-fixation dressing with surgical waste. It has persisted as such with good function and stability for eight years.

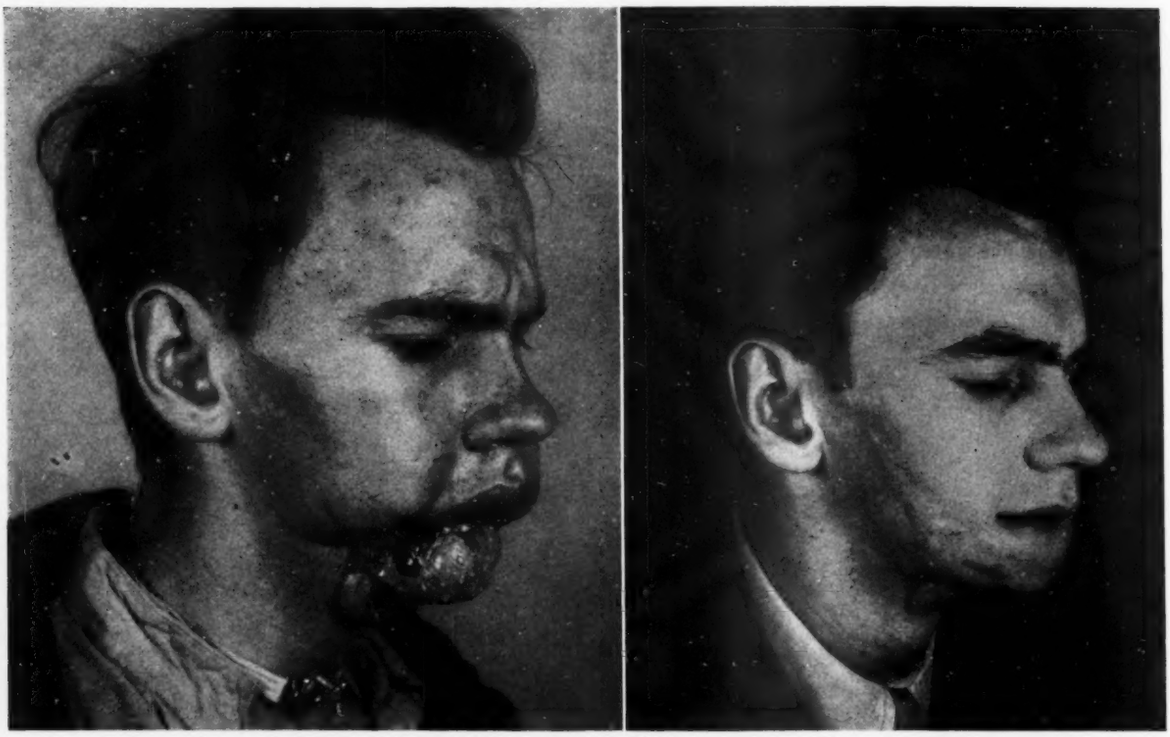


FIG. 4. Profile of injury and of final repair showing chin held forward by the bone graft. Flap in place; mouth watertight.

and supported with a waste pressure dressing. The bone graft is shown in Figure 3 eight years after operation. The jaw is solid, the wires are in place and it is thought that the bone graft has persisted as such.

The final result in Figures 1 and 4 indicates that the patient has made fine adjustment as seen in his expression. He is a trusted school board employee and has doubly earned the right to be considered as his own individual self, instead of just another "case" of gunshot injury of the face and jaw.

*Acknowledgment:* The patient was cared for at Valley Forge Hospital in association with Drs. B. Cannon, C. E. Lischer and A. M. Moore; Virginia McCall, artist; Ken Hoser, photographer; D. Saunders, W. Reed and Matilda Katterhenry, anesthetists; and H. Miller, L. Peterson and W. Smith, dental service.

REFERENCES

1. BROWN, J. B. and CANNON, B. Repair of major defects of the face. *Ann. Surg.*, 126: 624–632, 1947.
2. BROWN, J. B., FRYER, M. P. and MCDOWELL, F. Application of permanent pedicle blood-carrying flaps. *Plast. & Reconstruct. Surg.*, 8: 335–340, 1951.
3. BROWN, J. B., FRYER, M. P. and MCDOWELL, F. Permanent pedicle blood-carrying flaps for repairing defects in avascular areas. *Ann. Surg.*, 134: 486–494, 1951.
4. BROWN, J. B., CANNON, B., GRAHAM, W. C., LISCHER, C. E., SCARBOROUGH, C. P., DAVIS, W. B. and MOORE, A. M. Direct flap repair of defects of the arm and hand. *Ann. Surg.*, 122: 706–715, 1945.
5. BROWN, J. B., CANNON, B., GRAHAM, W. C. and DAVIS, W. B. Restoration of major defects of the arm by a combination of plastic, orthopedic and neurological surgical procedures. *Plast. & Reconstruct. Surg.*, 4: 337–340, 1949.
6. BROWN, J. B., PETERSON, L., CANNON, B. and LISCHER, C. Ankylosis of the coronoid process of the mandible (and associated scar limitation of jaw function). *Plast. & Recon. Surg.*, 1: 277–283, 1946.

# ACCELERATED EXCISION AND GRAFTING OF SMALL DEEP BURNS*

FRANK MCDOWELL, M.D.
*St. Louis, Missouri*

MANY deep burns which are not too large can be grafted within a few days after occurrence and the patients can be back at their normal routine within thirty days after the accident.

1A 1B 1C

FIG. 1. A, gasoline burn of leg first seen after one week; excision of burn next day. B, split grafting two days later; total of twelve days' hospitalization. C, back in school walking one month after burn.

Continuing efforts are being made to keep these deep burns out of the chronic category and in one in which recovery within a few weeks is the rule. Early grafting will minimize the time of disability and suffering, the amount of residual disability and the total amount of medical care and hospitalization. (Figs. 1 to 4.)

Superficial burns, however widespread, will heal within one to two weeks under kindly cleansing care and fine mesh grease gauze pressure dressings. Massive deep burns present serious, difficult problems in each case but do not occur in large numbers. Numerically, a major problem is that of the best care for the large number of small deep burns that are commonly seen.

Under consideration here are those deep burns that are large enough definitely to require grafting but not so large as to give cause for concern about the patient's general condition. These include deep burns of parts of extremities such as a hand, leg, forearm or similar areas in size elsewhere on the body.

If these burns are allowed to become chronic, prolonged granulation and inflammatory infiltration will occur with an increase in the resultant deep fibrosis and residual disability. There may be serious wastage of the patient's time and resources, but most important is any prolonging of pain and discomfort.

## IMMEDIATE EXCISION OF BURNS AND SKIN GRAFTING

On first thought immediate excision of these burns and immediate skin grafting would seem to be the ideal solution. This has been suggested by Wilms (1901), Murat Willis (1924), Wells (1929), Young (1942), Cope et al. (1947) and others. Actually, its application is quite limited although there is a definite place for it.

The first problem here is in judging the

* From the Department of Surgery, Washington University School of Medicine, St. Louis, Mo.

2A 2B

FIG. 2. Hot water scald of both thighs; burns excised one week later. A, split grafting few days later; B, home walking one month after burn.

correct depth of the burn in all of its parts. This is fairly obvious in some burns but is not in many others, especially if the observer has watched a large number of burns throughout their healing period. Even if it is definite that the full thickness of the skin has been burned through, it may not be clear just how much of the subcutaneous tissue is damaged beyond recovery. Small burns in fat areas such as the buttocks, thighs or abdominal wall can be treated in this manner as it is always possible to excise the burns and then cut out a little more fat until one is sure that a viable bed is present before applying the graft. This is especially applicable in small deep burns from molten metals or electricity. Thin areas such as the dorsum of the hand, nose and eyelids allow so little working margin that one should be cautious about undertaking this procedure in these areas.

The advice to solve this problem by excising all questionable areas and grafting them is not good. It may be good from the standpoint of economy or of an overbusy surgeon, but it is not good from the standpoint of the patient, as grafted skin is seldom as good as superficially burned skin that has regenerated satisfactorily.

The difficulty of judging the depth of the burn into the subcutaneous tissues, plus the initial edema under burned areas, has made the "take" of grafts somewhat uncertain when applied immediately. In some reports the percentage of "takes" has been as little as 60 per cent, with more than a few requiring secondary grafting.

Large burns should not be treated by this method, as the mortality figures will show if it is persistently used. The patient must be able to withstand his original burn shock, the shock of denuding another area at least as large for grafts, the shock incident to further bloodletting from the burn and the donor area, and the long anesthesia necessary.

Despite these objections immediate excision and immediate grafting is worth while in some small burns as noted heretofore.

### ACCELERATED EXCISION OF BURNS AND SKIN GRAFTING

Although immediate excision of burns and skin grafting has the disadvantages just noted, long delays are also to be avoided; and attempts have been made to find some satisfactory middle course. Spontaneous autolysis or separation of the slough may require as much as two weeks or more and this problem has not been solved with the various agents for chemical débridement that have been tried.

The surface area and the depth of these burns is usually demarcated quite well in from two to four days, so that accurate surgical

FIG. 3A. Fire burns of thigh, elbow and hand; excised four days after accident and grafted three days later.

FIG. 3B. Home walking and healed twenty-one days after the burn.

excision is then possible. At the time of excision there may still be some edema of the subcutaneous tissue or even liquefaction of some fat, so that grafting is best delayed for a day or two. Even so, grafts are usually in place and growing before the first week is ended, and within three or four weeks healing is often stable enough that bandages may be omitted except over dependent areas.

The general method of care is as follows, with variations as indicated: Patients with burns of this extent are usually first seen as outpatients. The burned areas and surrounding skin are cleansed with soap and water, and wrapped in occlusive, fine mesh grease gauze, pressure dressings. Such splinting as is indicated is done and they are instructed to keep the involved areas at rest and elevated at home. Antibiotics are often administered. When the subject is an outpatient, the first dressing is done on about the second or third day. If the burn is definitely deep and demarcated at that time, the patient is hospitalized and the deeply burned area is accurately excised with a scalpel, using general anesthesia. There is usually no difficulty by this time in ascertaining the depth. Bleeding is checked by pressure with weak adrenalin sponges or warm saline compresses. Following this the wound is flushed with saline and a wet saline pressure dressing is applied, with fine mesh gauze next to the wound. The following day the dressing is changed down to the fine mesh gauze and another wet saline pressure dressing applied. The second day after excision the patient is taken back to the operating room, the dressing removed and the wound washed gently with saline. At this time there will usually be a fine, thin skein of bright red granulations just visible. If so, split grafts are cut and applied over these granulations; it is an advantage to cut a single graft large enough to cover the entire wound when possible. The grafts can usually be just wrapped or "snubbed" on smooth surfaces, but suturing is worth while on irregular areas such as the hands.

If granulations are not present two days after

4A 4B

FIG. 4. Fire burn of leg. A, first seen later because of original uncertainty as to depth; burn excised a little later. B, permanent coverage with split grafts; total of eight days' hospitalization.

the excision, grafting is delayed another day or two, excising any further remnants of necrotic tissue that may be apparent. This delay, however, seldom occurs.

The first graft dressing is done in four or five days and thereafter as indicated. It is often possible to discharge the patient and to continue the care on an outpatient basis a few days later, especially with grafts on the upper extremities or body. Even with a leg graft, if there is a good solid take, it may be possible to wrap on a secure pressure dressing after nine or ten days and then change it on an outpatient basis at intervals of several days until no further protection is necessary.

Adults can often resume work or children resume school within one month after the injury; because of this it has sometimes been referred to as "the thirty-day treatment of deep burns." However, the duration of treatment will vary considerably from one patient to another.

It is thought that similar accelerated treatment of deep burns is being carried on in several places but knowledge of this is apparently not widespread, so that some description of it at this time may be helpful.

In large or massive deep burns, where fluid balance may be disturbed or even life threatened, such acceleration is not indicated. There will be less blood loss and general disturbance of the patient in these if the burn slough is allowed partially to separate spontaneously and is then removed by minimal dissection after ten days to two weeks. Grafting can then be done a few days later, sometimes in stages or even with temporary use of homografts for some areas.

The small or moderate-sized deep burn is probably the most frequent problem of all and it is hoped that this accelerated care for them will be of interest. The amount of hospitalization is reduced but variable, being determined by the condition and location of the grafts and by the patient's individual needs in each instance. The primary aims of the method are to shorten the period of pain, discomfort and disability, and to get the best possible final function by minimizing deep scar formation. However, patients and their families sometimes believe that the associated saving in hospitalization and time off from work is an agreeable by-product.

## REFERENCE

1. BROWN, J. B. and MCDOWELL, F. Skin Grafting, 2nd ed. Philadelphia, 1949. J. B. Lippincott Co.

# TREATMENT OF BURNS BY EXCISION AND IMMEDIATE SKIN GRAFTING*

ALFRED H. WHITTAKER, M.D.
*Detroit, Michigan*

THE first annual session of the American Association for the Surgery of Trauma, which the writer was privileged to attend, was the Atlantic City session of 1940. Dr. Glover[1] presented a paper on burns, the title of which was, "Fifteen Years' Experience with Tannic Acid Treatment." Since that time at almost every annual session some phase of burn treatment has been discussed.[11]

Some years after the end of World War II and after two years of war in Korea, burns continue, however, to be an unsolved problem in civilian life and industry. Considerable progress has been made in the care of the burned patient, particularly with the problems presented by the physiologic imbalance which develops following extensive burns, but there has been very little progress made in the treatment of the wound itself. This is emphasized by the numerous discussions which appear at various surgical meetings advocating various types of treatment.

An occasional surgeon has recommended the expeditious treatment of the wound, but observation reveals that the treatment as carried out in the hospitals of this country reflect the conventional methods which have been popular for many years, with changes from certain types of treatment, such as the paraffin treatment which was discarded, to be followed by the use of tannic acid which in turn, has been almost entirely replaced by the application of pressure dressings, of open air treatment or some other form of treatments which have actually been in use for a long period of time.

The suggestions to be presented in this paper deal with the full thickness burns, and particularly those which permit the immediate excision of the burn, or in the more extensive burns, an excision of the burn about the parts of the body where function is essential and covering of the resulting wound by split thickness skin grafts, or in some cases, pedicle grafts.

The review of the history of the treatment of burns reveals that Hipprocrates,[2] 450 years before Christ, advocated the application of various types of preparations, the treatment being entirely expectant in nature, waiting for the separation of the destroyed tissue and the application of various preparations to promote healing. Five centuries later Galen suggested much the same type of treatment, and Paulus Ægineta about 650 A.D. reviewed the methods of various physicians. He also referred to the methods of Hippocrates, and states that Dioscorides and Avicenna agree in praising various types of applications to recent burns and mentions the type of preparations that were in use.

Many centuries later Larrey, as a result of wide military experience, also advocated various forms of applications, and by 1829, Samuel D. Gross, translating "The Elements of Operative Surgery" by A. Tavernier,[5] divided burns into six types or degrees outlining a different method of treatment for each type. These treatments[4] consisted largely of the use of various astringent solutions and emolients, much the same type of treatment as that carried out by Hippocrates. Where the ancient surgeons had used animal preparations, Larrey advocated dressing the part with fine old linen spread with saffron ointment or honey, the separation of the eschar to be promoted by means of styrax ointment.

Summarizing the experience gained in World War I, De Tarnowski[6] lists various types of burns, and advocates paraffin dressings in third degree burns, and goes on to state that daily dressings are necessary, and that the presence of pus is not a contraindication to skin grafting and the sooner the latter is resorted to, the better will be the chance for the graft to live permanently. He further states that all deep burns of the face, neck and fingers should be grafted.

In 1941 Michael Mason[8] made a plea that

* From the Department of Surgery, Detroit Memorial Hospital, Detroit, Mich.

burns be regarded as surgical wounds and thought that results might be improved if the same principles used in the treatment of other wounds be followed. These principles, cleansing, débridement, hemostasis, closure and rest, are well known and generally accepted in the treatments of wounds, but the suggestion has not been applied to the treatment of burns. Moreover, it was pointed out that these rules hold only if the wound is cared for within six hours, certainly not longer than twelve hours. Referring to this type of treatment for burns, he suggests that the rules have not previously been accepted because of the wide extent of the burn injury at times and the poorly understood cause of the shock found in severe burns.

In 1942 Forest Young,[7] in a preliminary report, advocated the immediate grafting of full thickness burns. The method included a period not longer than six hours after injury, careful evaluation of the physiologic balance, parenteral plasma to be started, and under anesthesia the burn to be washed with soap and soft gauze, flushed with saline, and he advocated that no antiseptic be used. Obvious third degree burns were excised and split thickness grafts were cut with a dermatome and applied to the defects caused by excision of the burned tissue. Both donor and burn sites were dressed with vaseline gauze.

Tannic acid had been proven to be harmful, a contribution made by Dr. Roy McClure and the pathologist, Dr. Hartman, both of the Henry Ford Hospital and both having been associated with Davidson in his early work with tannic acid.

The partial thickness burns, which made up the largest number, were best treated by ignoring the wound and giving attention to the patient, a simple dressing being applied to the burn.

No improvement in the results of the treatment of full thickness burns occurred, however, largely because of the insistance that immediate attack on the destroyed tissue could not be carried out. The slough was allowed to separate out in a time-consuming procedure, infection always occurring, to be cleaned up later by wet dressings.

Chemotherapy helped by controlling infection, but the chemical could not penetrate the eschar, and even when blood levels were high they did not prove fully effective.

The practice of watching the eschar for a period of time as it cracks and becomes infected, about the fourth day, and finally separates, continues to be popular. The area of the burn, following an early stage of edema during the first few days, is followed by increased vascularity. Thus if delayed removal of the eschar is attempted, instead of one or two bleeding points which can easily be ligated, a delayed removal is carried out which results in bleeding, often in an important amount.

Frequently the slough is accompanied by a profuse discharge with loss of proteins and blood chlorides, systemic absorption of the burn products takes place and anemia develops with loss of healing power.

In 1947 Collumbine, McDonald and Simpson[12] made an important discovery which influences our thinking in the care of fresh burns. They discovered a polypeptide of low molecular weight which they called leukotaxine, obtained from the exudate of infected tissue which, when injected, produced an edema and blood changes similar to those found in burns. This product was only present after forty-eight hours.

During this same time the plastic surgeons were demonstrating that the fresh tissue wound bed was best for grafting, but the treatment of burns continues to be waiting for a "healthy granulating bed" to form, and grafting was carried out on the twenty-fifth day or later. In the meantime, fibrosis occurred, joints and tendons became frozen and the systemic and psychologic changes became irrevocably fixed.

In 1947 Cope[9] and associates advocated the expeditious care of full thickness burn wounds by surgical excision and grafting. After describing the nature of the deep burn and bacterial invasion occurring, they conclude that counter measures that intercept infection and lead to rapid healing must therefore include prompt removal of the necrotic tissue. If removal were immediate and closure of the wound accomplished by grafting, the inevitable contamination of the burn wound would be prevented from developing into an infective process. Since the invasion of bacterial organisms is rapid, the only means to eliminate the necrotic tissue before infection develops is direct surgical excision.

The elimination of infection is accompanied by improved nutrition, maintenance of physiological balance and prompt closure of the

wound results in minimal scarring, dsiability and disfigurement.

The early healing which takes place following grafting achieves a short period of hospitalization, an economy of manpower and a hopeful outlook for the patient.

In 1950 Miller and Posch,[10] in a paper dealing with burns of the hand presented before the American Association for the Surgery of Trauma, and with a wide experience in burns at the Detroit Receiving Hospital, stated that immediate grafting was not advisable, and other clinicians speaking before other surgical meetings were making much the same type of statement.

From the few references given above, however, it is noted that the possibility of immediate elimination of the deep burn wound had been recognized, but the few investigators expressing this belief have been unable to convince the surgeons of this country treating burns that such a method can be used with great benefit to the patient.

The reasons for the reluctance to accept the newer concept are that immediate excision is impossible because of the frequency of physiologic imbalance, inadequate skin available in extensively burned patients and an inability to judge the depth of the destruction.

In our experience during the past ten years it has been possible, except in very extensive burns, immediately to excise the destroyed tissue resulting from heat or the destruction of tissue taking place by direct trauma in the nature of a brush burn, and the removal of the destroyed tissue has helped in maintaining the physiologic balance, and there has usually been sufficient skin available to cover the functional parts of the body such as the hand, feet and joints, except in the case of the very extensive burns.

Experience permits of an accurate estimate of the depth of the wound. Parts of the body such as the dorsum of the hands, the front of the wrists and the malar region of the face are easily destroyed and a greater depth of burn can be expected in these regions. On the other hand, the buttock, thigh and chin require very deep burns to destroy all of the epithelial elements present because of the deep-seated hair follicles.

To determine the depth of a burn it is necessary to determine if circulation is present in the damaged tissues. In the completely destroyed skin the circulation is absent, as determined by an absence of change of color when pressure is made. Direct inspection of the depth of the burn is possible by dissecting free the edge of the burned area to expose the area of edema which is formed by the heat which produces the burn, forcing the tissue fluid into the layer of tissue immediately adjacent to the burned tissue. This layer of edematous subcutaneous tissue permits of easy separation of all destroyed tissue with minimal hemorrhage.

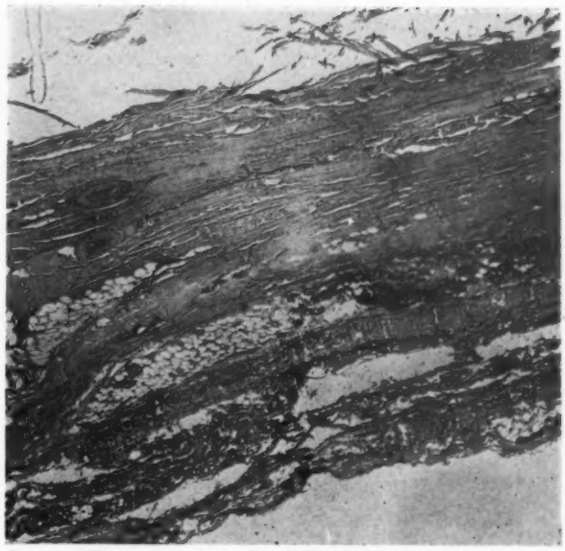


FIG. 1. Tissue section of a full thickness burn demonstrating the destroyed epithelium and the deep-lying, loosely arranged fragile edematous layer of tissue.

It should be remembered that in the excision of a burn when hemorrhage occurs even in rather large amounts, the loss is easily replaced by whole blood which has proved to be the best replacement agent.

If the burned area is limited in extent, the destroyed tissue may be excised radically; if extensive in nature, only those areas which are deep in nature and which occur in the region of a joint or other area of important function should be excised conservatively.

Experience has shown that for successful grafting all necrotic tissue must be excised, and no matter how deep the burn is, even involving bone, the defect can be closed by split thickness grafts.

Study of burns microscopically at various stages reveals that an eschar is immediately formed, which cracks with fissures occurring and that an area of edema is present immediately beneath the destroyed tissues. Bacterial invasion rapidly sets in so that by the

FIG. 2. Case 11. Photograph September 9, 1952. Full thickness burn extending to tendons. Entire boot of destroyed tissue excised day burn occurred and grafts applied day of injury.

fourth day frank infection is present. Increased vascularity rapidly develops leading to hemorrhage if excision of destroyed tissue is delayed. (Fig. 1.)

As time goes on a slough develops with infection, systemic absorption occurs, with increasing anemia, loss of protein and blood chlorides and the healing power of the burned area is impaired: With the separation of the slough a granulating bed develops which is not suitable for grafting, and during this period of delay extensive changes occur in the adjacent tissues with fibrosis and loss of function.

The picture is entirely different when immediate excision of all destroyed tissue is carried out. Prompt healing takes place with normal color of the skin covering the defect, and there is minimal contracture, very little discomfort and the psychologic attitude tends to be satisfactory.

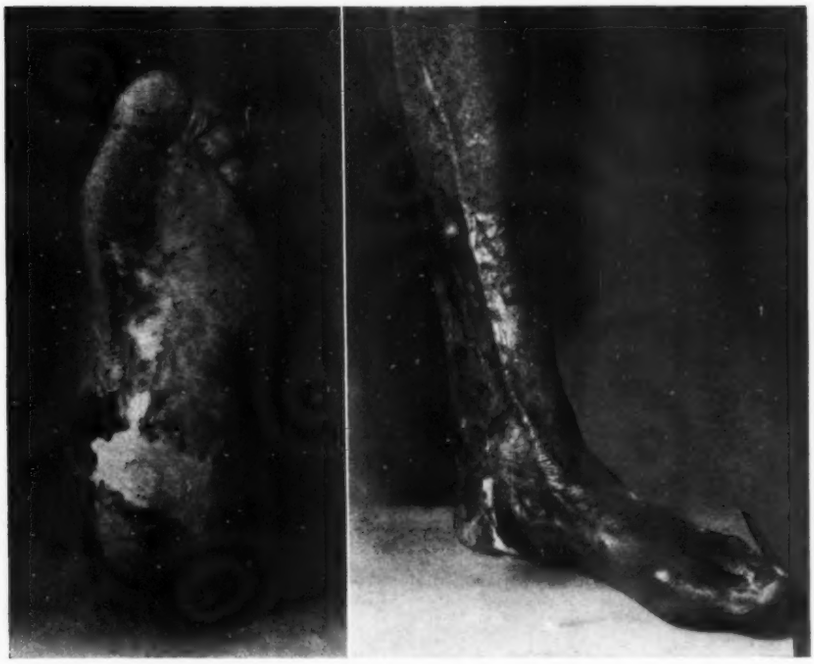
Starting in 1942 with smaller burns experience has gradually led us to excise rather extensive burns completely, within a few hours after the burn occurs, and in more extensive burns excising the destroyed tissue in the region of joints and about the neck. This immediate excision proves particularly valuable in the hands and feet. It has also been our experience that the best results have been obtained when the treatment has been prompt and carried out by a team composed of two surgeons and an assistant, an anesthetist who is familiar with the importance of the relationship of the physiologic balance of the patient to the extent of the surgery permissible, and a transfusionist with adequate whole blood available. (Figs. 2 to 6.)

The burned area is cleaned and irrigated as soon as the intravenous anesthetic is effective. A transfusion is started, and while one surgeon excises the destroyed tissue produced by the thermal agent, the second surgeon is preparing grafts by the use of a dermatome.

The operating surgeon excises the burn by separating the burn around its periphery which is the area which tends to be strongly fixed, and then removes the remainder of the destroyed tissue, following the loose edematous layer which lies immediately beneath the burned tissue. In the early excision a few bleeding points only are encountered and these are ligated.

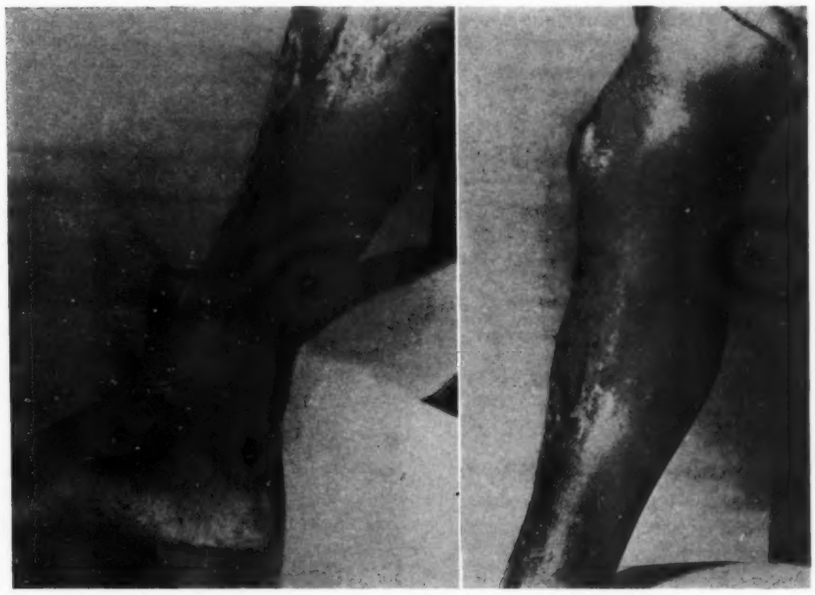
Care is taken to keep the grafts moist by use of saline and care is also exercised not to contaminate the donor site from the area of the burn. The surgeon removing the grafts hands the grafts to the surgeon who has removed the burned tissue and he and his assistant loosely suture the graft over the surgical defects resulting from the excision of the burned tissue, no effort being made to place them as an inlay, and incisions are made in the grafts to permit escape of blood from beneath the grafts. The grafted areas are covered with paraffin net and the grafts are held in contact with the underlying wound with an elastic pressure dressing consisting of fluffed gauze pressed upon by an ace type of bandage. Immobilization is insured by use of a plaster mold when the burned area involves an extremity. Great care is taken to place the part in a position of function.

The grafts are then not dressed for ten days unless infection develops, and any traction on



3 4

FIGS. 3 and 4. Case 11. Photographs September 9, 1952. Full thickness burn extending to tendons. Entire boot of destroyed tissue excised day burn occurred and grafts applied day of injury.



5 6

FIG. 5. Full thickness burn of the leg.

FIG. 6. Same Case as in Figure 5 demonstrating the end result of immediate excision of a full thickness burn involving the capsule of the knee joint. Skin grafting a few hours following the injury.

the grafts is avoided until they are firmly attached.

Full attention is then immediately concentrated upon the condition of the patient, the burns being intentionally neglected to permit every effort to eradicate the systemic effects of the burn upon the patient.

The illustrations which I present reveal the end results of immediate excision of deep burns about the hands and feet and various joints and the excellent functional results which can be obtained.

REFERENCES


1. Glover, D. M. A summary of fifteen years' experience with the tannic acid treatment. Papers of the American Association for the Surgery rauma, 1940.
2. Adams, Francis. Hippocrates, The Genuine Works of; printed for the Sydenham Society, London, 1849.
3. Adams, Francis. Ægineta, Paulus, The Seven Books of; printed for the Sydenham Society, London, 1846.
4. The House Surgeon and Physician, The Best American Remedies, Hartford, 1818.
5. Tavernier, A. Elements of Operative Surgery, translated by S. D. Gross, M.D. 1829.
6. De Tarnowski, G. Emergency Surgery, 1926.
7. Young, F. Immediate skin grafting in treatment of burns. *Ann. Surg.*, 116: 445, 1942.
8. Koch, S. Transplantation of skin and subcutaneous tissue in the hand. *Surg., Gynec. & Obst.*, 72: 12, 1941.
9. Cope, O. et al. Expeditious care of full thickness burn wounds by surgical excision and grafting. *Ann. Surg.*, 125: 1–22, 1947.
10. Miller, H. and Posch, J. L. Acute burn of the hand. *Am. J. Surg.*, 80: 784, 1950.
11. Hamilton, B., Schiller, C., Whiteside, J. H. and Randall, R. E. Use of ACTH and cortisone in surgery. *Am. J. Surg.*, 83: 374, 1952.
12. Columbine, H., McDonald, F. and Simpson, M. M. The role of leukotaxine in the production of anhydraemia of burn shock. *J. Path. & Bact.*, 59: 467, 1947.


## DISCUSSION OF PAPERS BY DR. MCDOWELL AND DR. WHITTAKER

Truman G. Blocker, Jr. (Galveston, Texas): I had not planned to discuss either of these papers, but I think something should be said from the standpoint of local excision of the burn. Since the work of Allen, Koch and Mason, the excision of burns has been advocated by a large group of individuals. I think those of us who have tried this procedure still believe it is of great value in localized burns in which one can distinguish between the unburned area and the deep burn area. When, however, we have a larger area, say 5, 10 or 20 per cent of the body surface, we are always surprised later in the course of the treatment to find how much of the burn is deep and how much of the superficial burn has healed around the edges. In my opinion, it is still very difficult to determine early what part of the burn is full-thickness, and only in those areas where function must be restored very quickly, such as the hand, do we believe that immediate excision and transplantation of skin is of any considerable value except, of course, in such localized acid or metal burns, as were demonstrated by Dr. Whittaker.

Dr. Brown's discussion has emphasized one of the most important things that have been advocated in the last few years, I think, the development of plastic and burn centers. The bringing together of individuals who are well trained in reconstructive work is of great value, particularly in time of disaster. The only disadvantage that I know of in such institutions would be the collection of pathogenic bacterial flora in the burn center. However, the bacteria cultured in almost all hospital infections are more innocuous than those that are found out in small, isolated hospitals. For instance, in our own center at the University of Texas we rarely see a streptococcal infection except in chronic burn cases on arrival. After a week's time, regardless of the type of antibiotic therapy we employ, the bacterial flora of our own service has become so antagonistic to the streptococcus that it has disappeared. The majority of our chronic cases have a staphylococcal infection which is resistant to penicillin but is of lower pathogenicity. Rehabilitation of burn patients by forced feeding and by the maintenance of high moarle with individual attention and the interest of the entire staff occurs much more rapidly in burn centers than in an institution where only an occasional severe case of burns is treated and where the burden of care is almost too much for one doctor to handle.

Harry E. Mock (Chicago, Ill.): I agree with you that these two papers are too good and too important not to receive considerable discussion. In the first place, I want to tell Dr. Brown that he is entirely too modest when he says these are not very good results. Those of us who have visited Dr. Whittaker and have seen his work in Detroit cannot help but feel that his advocacy of this method of dissection and early skin grafting is worthy of considerably more thought and attention by all of us.

My chief reason for getting up is to mention the fact that in October, 1917, when I was ordered off evacuation hospital No. 1 seven days before we were to sail, I was put in the Surgeon General's office as Assistant Director of Physical Reconstruction and Rehabilitation. I was fit to be shot. I had never heard of rehabilitation or physical reconstruction. They were new words in our nomenclature at that

time. Today in almost every paper we hear rehabilitation mentioned, showing how rapidly we have advanced from those days when too many of us thought only of the surgical end result and not of the functional result and the future economic and social life of the individual. I just want to praise you for the humanitarian words that you used, showing the extent we have gone since 1917.

Col. William H. Amspacher (Fort Sam Houston, Texas): Although I can add little to the excellent work which has been presented, there are two or three points that I should like to make.

During the past year we have been sorely tempted to do primary excisions on a number of our burn patients. However, we operate in a Burn Center where most of our cases are referrals and no one has seen fit to send us burns of limited extent such as those shown by Dr. Whittaker.

A very high non-protein nitrogen and relatively normal blood urea nitrogen develops between the fifth and the tenth day in a certain percentage of the patients who have deep, extensive burns. Most of the non-protein nitrogenous fraction has never been identified and the source is not known with certainty. It is believed, however, that the unidentified portion originates in the devitalized, burned tissue and that early excision of this tissue might eliminate the source of difficulty. We have hesitated to do a complete primary excision of the extensive burn because often not enough skin remains to cover the denuded area and we have not known exactly what to do with the resultant large open wound. I should like to hear from Dr. Whittaker concerning his experiences with these more extensive burns.

With reference to the paper by Dr. McDowell, I should like to reemphasize what has been said about gentleness in the care of the burned area. Chemical gentleness is just as important as physical gentleness, and any substance applied to a burn wound which is not completely bland can only add insult to the already injured tissues.

We are obtaining increasingly good results in burned hands. These results are attributable to better positioning, early skin coverage and immobilization for a period of time. In the young adult we do not hesitate to keep a hand immobilized in a good position until a partial thickness burn is completely healed or until a full thickness burn is excised and grafted. The period of immobilization may be twenty-one days or longer. A large, firm dressing over the hand is most important during the period after excision and grafting. These dressings should be applied until skin coverage is obtained. The dressing also is beneficial as an aid in producing a soft, non-edematous hand. We have seen many hands that have been ruined by overzealous attempts to secure motion in infected, thick, painful, uncovered, edematous extremities.

Isidore Cohn (New Orleans, La.) I heard the statement made that our improvement is largely due to the establishment of burn centers. You may have all the centers you please to no avail if you do not have leadership and humane individuals like Barrett Brown to head those centers. Anyone who has had the privilege of seeing Barrett Brown treat wounds at Valley Forge will understand and appreciate what the human and the humane element did. I remember on one occasion visiting with him when he had an aviator whose eyes had been lost. His whole face was covered, and when Barrett touched his hand the man said, "Dr. Brown, I am so glad to see you."

We need more leaders, more humane treatment, and less attention to mechanical procedures.

Alfred H. Whittaker (closing): I was asked one question and I shall try to answer it briefly. In the more extensive burn, where do you get the skin? There are only two places that skin is available. One is from the patient, and the other is from some friend or member of his family. They both can be made use of but, of course, there are many burns that are so extensive that there is no question of immediate grafting. For instance, I have photographs of people whose whole bodies were just a charred crisp of carbon and in such cases there is no question; but there are patients who come within the realm of immediate treatment and those are the ones I was talking about today.

There is one thing I think we can keep in mind. There are many burns in which we can cover the whole area immediately, and there are other burns where the area involved is rather extensive. We do know that if we wait until these slough and then heal, and graft later, we are going to get a fibrosis about the functional area, and we are going to get a freezing of the joints perhaps, and permanent disability. There are some cases even in which the burn is rather extensive that we can do a localized grafting about the joint areas and leave the rest of the burn for future reference.

If the burn is so extensive that it cannot be grafted immediately, it becomes a problem of general burn treatment, in which we have to consider the physiology of the patient rather than the wound itself. The wound is covered only temporarily in a simple way and forgotten, and attention is paid to the patient himself.

# RUPTURE OF BLOOD VESSELS BY STRENUOUS PHYSICAL EXERTION*

CHESTER C. GUY, M.D. AND W. A. WICHOWSKI, M.D.
*Chicago, Illinois*

IT is well recognized that a blood vessel with walls weakened by disease may rupture spontaneously or during physical exertion which temporarily elevates blood pressure. Such examples as rupture of aneurysms of the aorta or basilar cerebral vessels are too well known to merit more than passing comment.

We became interested in a study of those cases in which definite physical exertion has caused or been closely associated with the rupture of other blood vessels, with resulting formation of a hematoma. With one exception comparatively little discussion is found on this subject, even in books devoted to the relationship of trauma to disease. The exception is the rupture of the inferior or deep epigastric artery producing a hematoma between the rectus muscle and its posterior sheath or peritoneum. A classical example was seen by one of us at the Cook County Hospital.

A forty-six year old white woman was carrying a heavy basket of wet clothes when she suddenly tripped and maintained her balance only with difficulty. She felt an immediate pain in the abdomen which was relieved in part by flexion of the body and thighs. She became nauseated and vomited once. On admission to the hospital she was bent over and resisted all attempts to extend her legs. The abdominal muscles were held in board-like rigidity and maximum tenderness was located over the right rectus muscle below the umbilicus. The temperature, pulse and leukocyte counts were normal. There was no significant past history other than that she was the mother of eight children. On the following day a poorly outlined mass could be felt in the region of maximum tenderness. The correct diagnosis was not made preoperatively although the history and findings were classical. Through a low paramedian incision a large hematoma was found between the right rectus muscle and peritoneum. (Fig. 1.) No tears in the muscle were identified and the exact site of rupture of the epigastric vessels could not be found. The hematoma was evacuated and the vessels tied after excision of segments of both arteries and veins. She made a prompt and uneventful recovery. Microscopic sections of the vessels revealed no abnormalities.

Rupture of the epigastric arteries was first described by Richardson[1] in 1857. Maydl[2] in 1882 and Wohlgemuth[3] in 1923 surveyed the literature on the subject and many other articles have appeared since the latter date. At least 165 cases have been reported. Of the 127 reviewed by Wohlgemuth, seventy-nine were below the navel and eighteen below the semilunar fold of Douglas. Premonitory soreness of several days' duration often is a complaint preceding the acute severe pain of the more extensive hemorrhage. A mass of varying size gradually appears which is confined usually to the rectus sheath as emphasized by Fothergill.[4] Below the fold of Douglas there is no posterior sheath and the hematoma may extend extraperitoneally into the pelvis and be palpable on vaginal examination. If rupture of the hematoma occurs through the peritoneum, the mass may disappear, as in the case described by Brindeau.[5]

It is obvious that this entity must be differentiated from a variety of conditions producing the picture of an acute condition of the abdomen.[3,4] The correct diagnosis should be made if the condition is considered but it is usually confused with incarcerated hernia, tumors or infections of the abdominal wall, torsions of an ovarian cyst, intestinal obstruction and perforations. In a slowly developing case Laffont[6] noted discoloration around the umbilicus. Hilgenreimer[7] had seven cases, none correctly diagnosed preoperatively. Koerte[6] thought his patient had gallstone colic. Behan[8] diagnosed one case as acute appendicitis and one as incarcerated hernia. Maxwell[9] reviewed fifty-seven cases and found that the diagnosis was incorrect in forty-seven of them, and the actual

* From the Surgical Service, Illinois Central Hospital, Chicago, Ill.

pathologic condition was suspected in only nine. Culbertson[10] noted the infrequency with which this disorder is considered, even in textbooks of gynecology. It occurs much more frequently in women than in men, and pregnancy is an important factor. The onset of the hemorrhage is usually associated with some physical exertion although this may be of mild degree. Robertson[11] and others have questioned whether the rupture of blood vessels is primary or follows a primary rupture of the rectus muscles.

Probably the next most common site of blood vessel rupture with physical exertion is in the lower extremities. We have seen a considerable number of these and the following is a good example:

A sixty year old man had no known circulatory disturbances of his legs and no obvious varicose veins. During a golf game he took a particularly vigorous swing and felt a sudden pain along the posteromedial aspect of the right thigh. A few hours later he was aware of a tender, hard swelling which gradually increased to the size of a tennis ball. He continued his daily activities and reported to us five days later when he noted hemorrhagic discoloration extending down the medial aspect of the thigh to below the knee. (Fig. 2.)

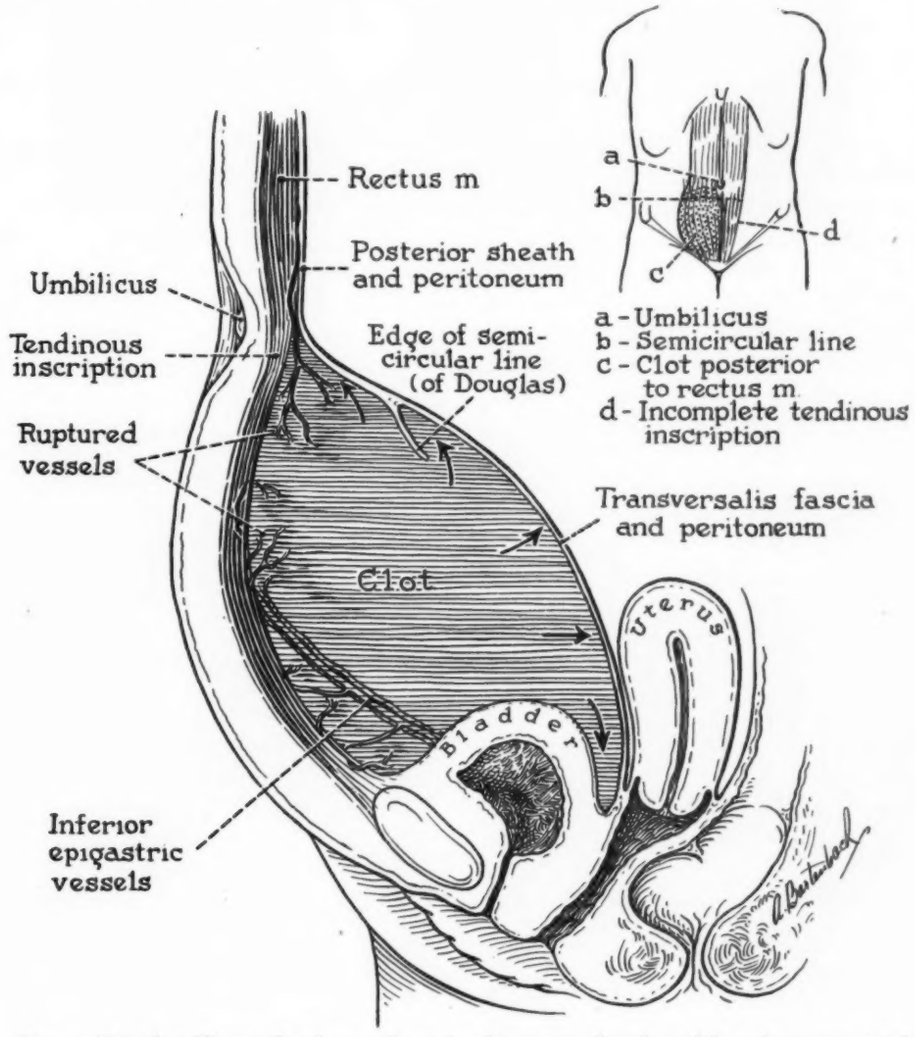
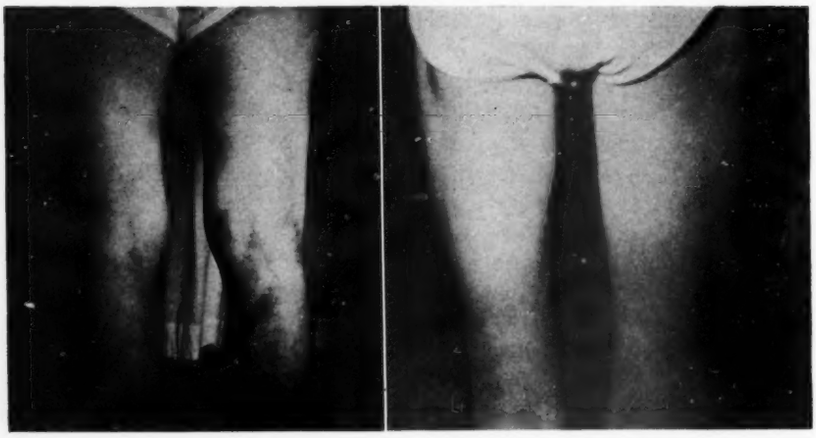



FIG. 1. Drawing illustrating hemorrhage in the rectus sheath and how it may extend into the pelvis.

We have noted on several occasions that the diagnosis of a ruptured blood vessel is not considered until skin discoloration appears, often several days after the onset of the disability, and occasionally after the major disability has ceased. This has been noted in the low back,



2 3

FIG. 2. Hemorrhage in skin of leg and thigh resulting from vigorous swing of a golf club.

FIG. 3. Swelling of right thigh following strenuous exertion; originally diagnosed as muscle strain but later proved to be due to hemorrhage.

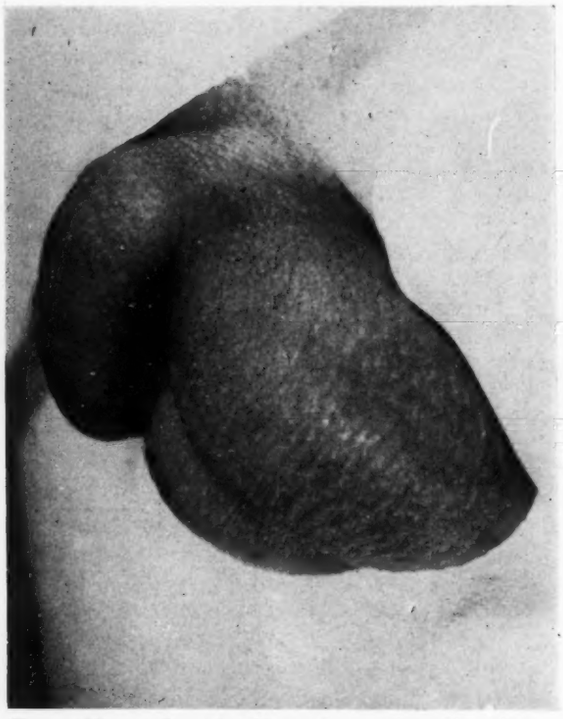


FIG. 4. Hemorrhage in skin and scrotal swelling due to hematoma following lifting of heavy weight.

FIG. 5. Hematoma after dissection from cord and testicle.

the calf of the leg, on the abdominal wall, about the shoulder girdle and on the inner aspect of the upper arm. Most of these have been originally diagnosed as muscle strain, and the significance of possible deep formation of a hematoma not appreciated. (Fig. 3.) Some of the cases diagnosed as thrombosis of the subclavian or axillary vessels are undoubtedly hematomas from ruptured vessels due to exertion. We have seen one such patient in whom improvement promptly followed evacuation of a hematoma surrounding the lower axillary vessels.

An unusual case was that of a sixteen year old white boy who was employed as a mail clerk. He lifted a large sack of mail, weighing about 100 pounds, and deposited it on a rack higher than his head. With this exertion he felt an immediate severe pain in his left groin, a sensation of "something being torn loose," and was sent to the hospital. Tenderness and

slight swelling were noted in the inguinal region and conservative treatment prescribed. By the next day there was a faint discoloration of the skin at the root of the penis and upper scrotum, with scrotal swelling and tenderness. (Fig. 4.) A diagnosis was now made of torsion of the testicle. At operation a large hematoma was found in the inguinal canal extending down the cord and surrounding the testicle and epididymis. By careful dissection this mass was freed and removed, preserving the spermatic cord and artery. (Fig. 5.) There were no evidences of hernia or varicocele and no circulatory changes in the testicle. Examination one year later revealed normal structures with no testicular atrophy. Careful study at operation and of microscopic sections of excised segments of the pampiniform venous plexus failed to reveal any evidence of blood vessel disorder. A pre-employment physical examination of this young man one month prior to the accident had shown no abnormalities. The patient and witnesses stated that there could not have been any direct blow to the inguinal area. We believe this case is unquestionably one of rupture of apparently normal blood vessels by violent muscular effort.

Many illustrations can be given of ruptures of blood vessels following physical strain in various parts of the body. Rupture of nasal and conjunctival vessels from violent coughing is an example. Cushman and Kilgore[12] described a syndrome of mesenteric or subperitoneal hemorrhage, reviewed the literature and reported four cases. In their survey three patients were found to be under forty years of age and one was only twenty-seven. Arteriosclerosis was demonstrated or presumed in four-fifths of these patients and well marked hypertension was present in one-third of them. On exploration of seventeen patients, in seven the source of the hemorrhage was found and ligated with one death. In the other ten no source could be found and four died. The over-all mortality was 30 per cent. Reimann and Cowley[13] could not find the bleeding point in 30 per cent of the patients explored in their series. Marks and Freedlander[14] estimated the surgical mortality as 26 per cent in abdominal apoplexy. Bunch and Madden[15] found that arteriosclerosis and hypertension were commonly associated with spontaneous intra-abdominal hemorrhage.

Payne[16] reviewed case reports from foreign literature and found that military surgeons of France, Germany, Austria and Russia have recorded cases of ruptured blood vessels following vigorous muscular effort of young males in military training. He concluded that tears of small blood vessels usually produce slow-growing tumors and a gradual exacerbation of symptoms, whereas tears of large vessels produce a rapid development of symptoms with acute and definite pathognomonic signs.

Koch[17] described a rupture of a subclavian artery due to an athletic strain. No disease of the vessel could be found at autopsy. St. John, Scudder and Stevens[18] reported a ruptured axillary artery due to physical effort. These authors and Koch suggest the possibility of some abnormal development of these vessels as a cause of the rupture, although it is admitted that congenital hypoplasia of arteries has been described only in the aorta and cerebral vessels. Longland[19] thought that congenital hypoplasia was the explanation of the spontaneous rupture of a pulmonary artery in his patient.

Payne[16] believes that some degenerative change in the blood vessels must be a frequent contributing factor as in the cases of rupture of the epigastric arteries. Herman[20] explains these cases by calling attention to the length of the epigastric vessels and their close attachments to the tendinous inscriptions. An incomplete inscription may occur in the lower third of the rectus muscle and is illustrated in Figure 1. With alternate contraction and relaxation of the rectus muscles, a rapid and relatively enormous change takes place in muscle length. The blood vessels, tightly bound to the muscle at intervals and loosely bound elsewhere, are unable to accommodate to the rapid change and are torn apart by a "crack of the whip" action. Thus the rupture is not "spontaneous" but follows a definite muscular action.

Illingsworth and Dick[21] state that rupturesof the epigastric vessels are more often due to muscular exertion than to direct violence, especially in pregnancy, parturition and debilitated states.

Payne,[16] Chalier and Vallery,[22] and Kenwell[23] have all discussed the influence of an infectious process in the body on rupture of blood vessels. Several case reports have to do with hematomas of the rectus muscle in patients with influenza. Culbertson[10] quotes Cooper Cole's observations of degenerative changes in the rectus muscles of patients with influenza. Klotz[24] found similar

changes in the pectoralis, psoas, gluteus and sternocleidomastoid muscles. Beals, Blanton and Eisendrath[25] found hematomas in the rectus sheath in eight of 140 patients who came to autopsy after death from pneumonia. They thought that localized infection in the rectus muscle with early abscess formation was followed by muscle rupture and hemorrhage incident to violent coughing. Sloan[26] reports that spontaneous hemorrhage in the rectus muscle was well known during the influenza epidemic in 1918. Many of these patients were operated upon for appendicitis. Bonnet, Martin and Nikodievitch[27] believed that atheromatous changes in the media plus the effects of infection caused the weakening of arteries predisposing to rupture. St. John, Scudder and Stevens[18] speculate on some vascular toxin with particular affinity for the media. Lasch[28] describes hemorrhages in pertussis due to actual changes in the walls of blood vessels which he ascribes to some toxic or infectious origin. Kaufmann[29] and Longland[19] discuss toxic or bacterial damage to walls of veins and arteries. Behan[8] found degenerative changes in the muscle fibers of the artery involved in his case. Klotz and Simpson[24] also describe degenerative changes with necrosis in the media as a prelude to rupture. Vitamin A and C deficiencies are suggested as possibilities by some authors (With,[30] Longland,[19] Opper[6]) while others (Lehman,[6] Malpas,[31] Norgaard[32]) ascribed cases to various blood dyscrasias. Still other authors could find no pathologic change to explain ruptures of vessels or muscles. Jaschte and Meldolesi[6] noted arteriosclerotic changes in the epigastric vessels, especially after repeated pregnancies. Dencks[33] examined the epigastric vessels and rectus muscles of ninety-five cadavers of both sexes, ages fifty to ninety. He could not identify enough pathologic changes to explain spontaneous rupture. From these and other varied and conflicting reports having to do with the etiology and pathology of ruptured vessels, it becomes clear that strenuous physical exertion can and occasionally does cause tearing of arteries and veins which are normal as far as can be determined. It is also entirely obvious that many vessels weakened by pre-existing disease are susceptible to rupture, even from mild exertion. From a medicolegal viewpoint, it therefore cannot be assumed necessarily that a ruptured vessel is or is not a diseased one. In the cases of rupture of the epigastric vessels, the most common type, there is usually no conclusive evidence of disease and there is usually a definite history of some physical exertion immediately preceding the disability, as in thirty-nine of forty-six cases reviewed by Maxwell. We now have methods for the study of the integrity of the circulatory system in various parts of the body. The x-ray, electrocardiogram, oscillometer, thermocouple, and other instruments and tests may be of value. Such studies are indicated in patients with hemorrhages anywhere following physical exertion and certainly should be made before giving testimony regarding the relationship of physical strain and ruptured blood vessels, unless disease of the blood vessels is clearly demonstrated.

## CONCLUSIONS

Rupture of normal blood vessels due to strenuous physical exertion apparently may occur. It has not been proven that pre-existing disease is necessary for rupture of vessels during strain and in the absence of direct violence.

The most common example is rupture of the epigastric vessels, usually with physical strain, in women who are or have been pregnant. Numerous other types occur, and if mild the diagnosis is easily missed. Such cases may have important therapeutic and medicolegal aspects.

## REFERENCES

1. Richardson, S. B. Rupture of the right rectus abdominis muscle from muscular efforts—operation and recovery, with remarks. *Am. J. M. Sc.*, 59: 41, 1857.
2. Maydl, K. Ueber subcutane muskelund sehnenzerreissungen sowie Rissfracturen, mit Berucksichtigung der analogen durchdirecte Gewaltenstandenen und offenen Verletzungen. *Ztschr. f. Chir.*, 17: 306, 1882.
3. Wohlgemuth, K. Subcutaneous rupture of rectus muscle. *Arch. f. klin. Chir.*, 122: 649–654, 1923.
4. Fothergill, W. E. Hematoma in abdominal wall simulating pelvic new growth. *Brit. M. J.*, 1: 1941–1942, 1926.
5. Brindeau, A. Hématome du muscle grand droit de l'abdomen chez une femme enceinte. *Gynéc. et obst.*, 30: 168, 1934.
6. Quoted by Payne.[16]
7. Hilgenreimer, H. Das spontane Bauchdeckenhämatom des vorgeruckten Lebensalters ein Breitrag zur Spontan-Ruptur des M. Rectus Abdominis. *Beitr. z. klin. Chir.*, 129: 700–710, 1923.
8. Behan, R. J. Rupture of rectus abdominis muscle. *Boston M. & S. J.*, 182: 660, 1920.
9. Maxwell, A. F. Spontaneous hematoma of abdominal wall in women. *California & West. Med.*, 30: 407, 1929.

10. Culbertson, C. Hematoma occurring spontaneously in sheath of rectus abdominis muscle. *J. A. M. A.*, 85: 1955, 1925.
11. Robertson, H. M. Hematoma of abdominal wall. *Canad. M. A. J.*, 36: 606, 1937.
12. Cushman, G. F. and Kilgore, A. R. Abdominal apoplexy. *Ann. Surg.*, 114: 672, 1941.
13. Reimann, D. L. and Cowley, R. A. Obscure causes of intra-abdominal hemorrhage. *Am. J. Surg.*, 71: 328, 1946.
14. Marks, M. and Freedlander, S. O. Spontaneous hemorrhage. *Ann. Surg.*, 121: 191, 1945.
15. Bunch, G. H. and Madden, L. E. Abdominal arterial apoplexy. *South. M. J.*, 34: 643, 1941.
16. Payne, R. L. Spontaneous rupture of superior and inferior epigastric arteries within rectus sheath. *Ann. Surg.*, 108: 757, 1938.
17. Koch, W. Rupture of an apparently healthy artery during athletic exertion. *Med. Klin.*, 24: 1473, 1928.
18. St. John, F. B., Scudder, J. and Stevens, D. L. Spontaneous rupture of axillary artery. *Ann. Surg.*, 121: 882, 1945.
19. Longland, C. J. Spontaneous rupture of pulmonary artery. *J. Path. & Bact.*, 55: 103, 1943.
20. Herman, C. Rupture of deep epigastric vessels. *Am. J. Surg.*, 71: 553, 1946.
21. Illingsworth, C. F. W. and Dick, B. M. Text Book of Surgical Pathology, p. 219. London, 1945. J. and A. Churchill, Ltd.
22. Chalier, J. and Vallery. Rupture musculaire du grand droit, hematome suppure à bacille d'Eberth au cours d'une fièvre typhoïde. *Lyon méd.*, 140: 493, 1927.
23. Kenwell, H. N. Spontaneous hematome of abdominal wall—case report. *New York State J. Med.*, 29: 1186, 1929.
24. Klotz, O. and Simpson, W. Spontaneous rupture of aorta. *Am. J. M. Sc.*, 184: 455–473, 1932.
25. Beals, L. S., Blanton, W. B. and Eisendrath, D. N. Abdominal complications of influenza. *J. A. M. A.*, 72: 850, 1919.
26. Sloan, L. H. Personal communication.
27. Bonnet, P., Martin, J. F. and Nikodievitch, V. Spontaneous rupture of arteries. *Lyon chir.*, 20: 566–582, 1923.
28. Lasch, W. Pathogenesis of hemorrhage in whooping cough. *Monatschr. f. Kinderh.*, 28: 441, 1924.
29. Kaufmann, E. Pathology for Students and Practitioners, vol. 1, p. 150. Philadelphia, 1929. P. Blakiston's Son & Co.
30. With, S. Hematoma of rectus abdominis muscle due to C avitaminosis. *Hospitalstid*, 80: 521–522, 1937.
31. Malpas, P. Spontaneous hematoma of rectus abdominis muscle with report of 2 cases. *Brit. M. J.*, 1: 1130–1131, 1930.
32. Norgaard, F. Spontane haematomer i mm. recti abdominis hos kvinder. *Hospitalstid*, 80: 246, 1937.
33. Dencks, G. Das spontane Bauchdeckenhämatom. *Deutsche Ztschr. f. Chir.*, 213: 159–169, 1929.
34. McKim, L. H. Partial rupture of the right rectus muscle with hemorrhage into its sheath. *Canad. M. A. J.*, 36: 181–183, 1937.

# ARTERIAL REPLACEMENT IN TRAUMA*

PRESCOTT JORDAN, JR., M.D., TOR HIERTONN, M.D. AND
CHARLES G. JOHNSTON, M.D.

*Detroit, Michigan*

THE need for a method of bridging defects in the major arteries in various injuries and diseases has long been felt. Although it was proved as early as the beginning of this century by Carrel[1] and others[2-4] that preserved arterial segments could be successfully used as grafts, the practical clinical use of arterial homografts was not demonstrated until recently.

The availability of arterial segments of different calibers and lengths, preserved in a blood vessel bank, appears to provide the surgeon with new and improved possibilities of treating not only congenital deformities of the heart and the major arteries, but also acute injuries and emergencies to the central and peripheral arteries.

It appeared from previous investigations that arterial tissue, removed aseptically within a few hours after death and refrigerated in nutrient medium, maintained its viability for days or even weeks. Opinions differ, however, about the importance of using such viable instead of non-viable grafts. It is still not known whether or not cells of a viable transplant do survive or whether they all die and are totally replaced by new cells from the host. If it is true that all cells of the graft are doomed to destruction, then the graft can be regarded simply as an organic tube or framework that the host cells use as a form. This method of replacement by new living cells from the host, which is not clearly understood, must be studied more carefully. Definitely devitalized arterial segments are also reported to be successful as homografts. As with the viable grafts, the non-viable may carry out their function in spite of marked histologic changes.

While the early results of arterial grafting have been good, there have been no adequate long-term follow-up studies. When we consider the histologic changes which occur early, it is conceivable that late complications may occur, i.e., a progressive degeneration of the graft, late thrombosis, rupture or aneurysm formation.

The use of autogenous vein grafts give a viable replacement which is now accepted[5] as the only type of arterial replacement whose cells do not perish in their new location. However, this type of graft does not completely answer the requirements of the ideal arterial replacement. In acute injuries or emergencies of the central or great arteries it is not applicable because of the disparity in size and, as Harkins[6] has shown, the late complication of aneurysmal dilatation. The work of Marrangoni and Cecchini[7] with lyophilized aortic segments interested us in this type of arterial prosthesis; because of its theoretically greater application in emergencies or acute traumatic injuries, coupled with its duration and simplicity of preservation, it seemed to be nearer the ideal arterial replacement. Although the idea is not original, as Carrel[8] dehydrated aortic segments and transplanted them experimentally, a complete investigation of the clinical and morphologic changes in this type of graft has not been reported. This study was undertaken to determine the fate and possible clinical application of such a graft.

## METHODS AND MATERIAL

These studies were made on two series of mongrel dogs, one group serving as donors and the other as recipients. The aortic segments were obtained using sterile technic and aseptic precautions were used in replacing segments into the recipient dogs. Bacteriologic studies of the segments were made and negative growths obtained in beef broth before segments were placed in the recipient's aorta.

The sterile arotic segments were obtained from the donor dogs and immediately placed into sterile test tubes and stoppered. These tubes were then immersed in liquid nitrogen

* From the Department of Surgery, Wayne University College of Medicine, and Veterans Administration Hospital, Dearborn, Mich. Aided in part by a grant from the Kresge Fund.

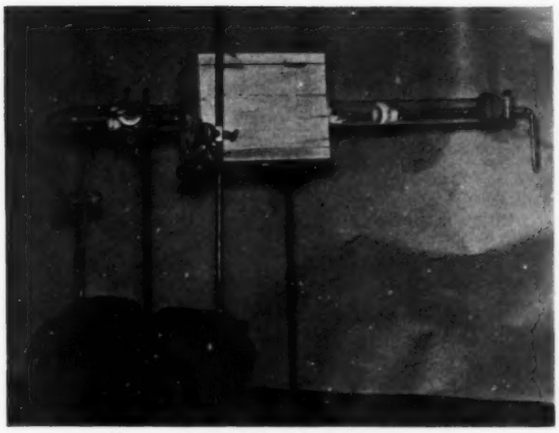


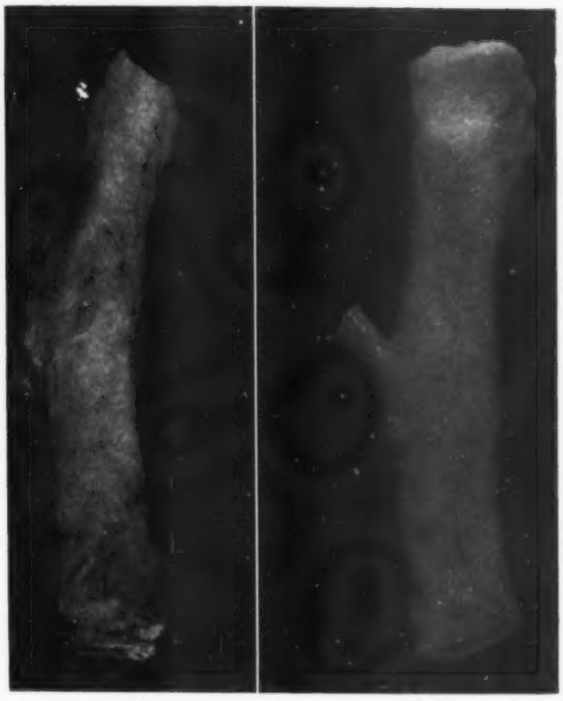
FIG. 1. Lyophilization apparatus.

for approximately ten to fifteen seconds and quick-frozen at −195°C. The frozen segments were then transferred to a bath of alcohol and crushed "dry ice" for transfer to the dehydrator. After thirty-six to forty-eight hours in the lyophilizer the segments were completely dehydrated and ready for use or storage.

The lyophilizing apparatus (Fig. 1) consists of a glass tube 2 inches in diameter and 2½ feet long with a box containing crushed solid $CO_2$ over the proximal one-third. The vacuum pump used was connected by a short hose with a glass petcock interposed between it and the vacuum tube. The tube on its distal end was also equipped with a glass petcock and tube. This simple lyophilizer was able to produce a negative pressure of approximately .05 cm. Hg (by measurement with a Zimmorle apparatus) without any signs of stress.

After the placement of a lyophilized segment in normal saline, it assumed a normal size and texture in about thirty to sixty minutes. (Fig. 2.) At this time it was pliable and, except for some decreased resistance to the passage of a needle through the wall, it resembled a fresh aortic segment. These segments could be stored either over calcium chloride or in hermetically sealed tubes left at room temperature.

After placement of these segments in recipient dogs aortograms were done in the postoperative period by retrograde technic. A small polythene tube was threaded up one of the femoral arteries and 10 to 12 cc. of either 70 per cent diodrast or thoratrast was used as a contrast media. As the dogs died the patency of these grafts was observed at postmortem examination and a segment obtained for histologic study and comparison with a similar



2A 2B

FIG. 2. Lyophilized graft, aortic. A, preserved graft; B, reconstituted graft after thirty minutes in saline.

preparation from the same lyophilized graft obtained before surgery.

Complete aortas were obtained from thirteen dogs and segments from the upper, middle and lower one-third of the thoracic aorta and the abdominal aorta were either lyophilized or preserved in Tyrode's solution. Patterns measuring 5 mm. by 10 mm. were then cut from these segments by means of a die. These patterns were cut both longitudinally and transversely to the long axis of these aortas. Using the method of Evans and Lebow,[9] the tensile strength and stress strain characteristics of these differently treated aortic segments were compared. These results were also correlated with similar studies of fresh canine aortas previously studied by one of us (T. H.).

## RESULTS

The results in the recipient group of ten dogs are summarized in Table 1. Of the four deaths which occurred, none were due to failure of the grafts. The death of the first dog (Table 1, dog 104) was due to a technical difficulty arising from the use of a thoracic segment in the abdominal aorta with a large discrepancy in the size of the lumens. The second death (Table 1,

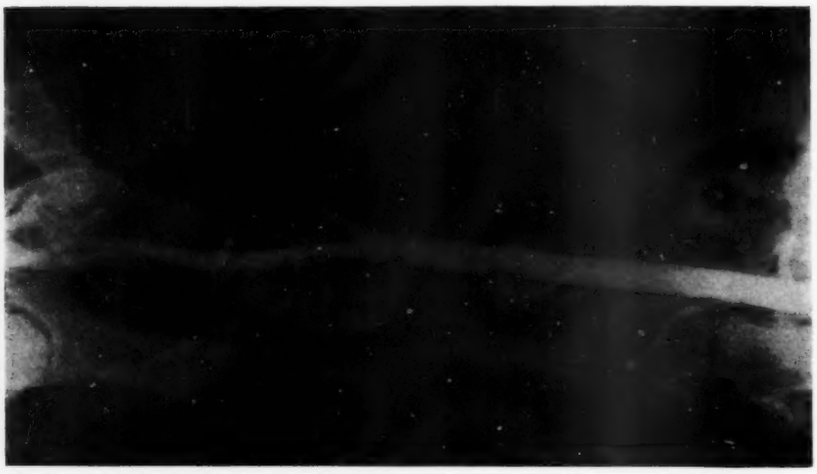


FIG. 3. Aortogram of dog 105 one month after graft was placed in aorta.

dog 108) was due to a leak at the suture line and hematoma formation which must be classified as death due to a technical error.

The remaining dogs have been checked by aortograph (Fig. 3) and/or arterial palpation and have shown open and patent grafts. Two of these dogs died due to causes unrelated to surgery, one dying fifteen days postoperatively (Table 1, dog 110) and the other (Table 1, dog 112) two months postoperatively. Both grafts were grossly patent without signs of aneurysmal dilatation or thrombus formation, the intima was smooth and glistening. (Fig. 4.)

Histologic studies carried out on these two dog's aortic grafts (Table 1, dogs 112 and 110) give no evidence of any unusual changes not seen in homologous grafts preserved in Tyrode's solution. There is a fibroblastic infiltration of the structures throughout the walls of the graft with a new intima-like cell layer growing down the tube from the host's intima. (Fig. 4.) Sections were also made from thirteen canine aortas which had been lyophilized. In these sections a combination elastic tissue stain and hemotoxylin-eosin method was used; thus in one section showing the normal architecture and elastic tissue pattern. These aortas were in part quick-frozen lyophilized using the liquid nitrogen method and the remainder were slow-frozen in alcohol and solid carbon dioxide.

TABLE 1

RESULTS WITH LYOPHILIZED AORTIC GRAFTS

| Dog | Operated | Aortogram | | Condition and/or Date of Death | Remarks |
|---|---|---|---|---|---|
| | | Date | Result | | |
| 104* | 3/24/52 | Not done | | Died, 3/31/52 | Ruptured distal suture line |
| 105 | 3/25/52 | 4/25/52 | Patent | Living (good distal pulse), 7/2/52 | |
| 106 | 3/26/52 | 4/23/52 | Patent | Living (good pulse), 7/2/52 | |
| 107 | 3/28/52 | 4/23/52 | Patent | Living (good pulse), 7/2/52 | |
| 108* | 3/31/52 | Not done | | Died, 4/8/52 | Breakdown of small area distal suture line with hematoma leading to death |
| 109 | 4/2/52 | Not done | | Living (good distal pulse), 7/2/52 | |
| 110* | 4/4/52 | Not done | | Died (graft good), 4/19/52 | Died of pneumonia, graft functioning, no dilatation |
| 111 | 4/8/52 | 4/25/52 | Patent | Living (good pulse), 7/2/52 | |
| 112* | 4/9/52 | 4/25/52 | Patent | Died in fight (graft patent), 6/17/52 | Graft excellent and widely patent, no dilatation |
| 113 | 4/9/52 | 4/25/52 | Patent | Living (good pulse), 7/2/52 | |

* These dogs died.

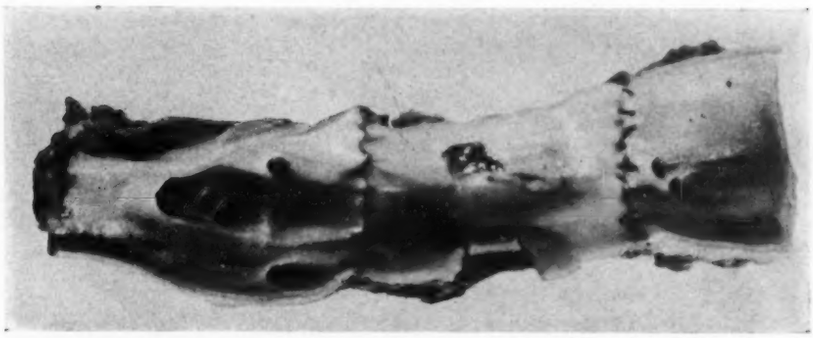


FIG. 4. Dog 112 two months postplacement of lyophilized graft *in situ*.

Those frozen quickly showed a normal architecture, while those more slowly frozen showed some foaming of the structures of the media. This histologic observation in the slow-frozen group was believed to be due to the formation of macrocrystals with resultant destruction of cell boundaries. Tissues which have been quick-frozen show no such cellular abnormality as the crystals formed are too fine to cause any cellular disruption. To protect the cells from disruption by macrocrystals the grafts were passed as quickly as possible through the (0°C.) to (−20°C.) range by quick-freezing in liquid $N_2$. On dehydration this temperature range was not thought to have been reached by the time the majority of fluid had been removed by evaporation.

The tensile strength studies are not completed. However, in preliminary comparison between fresh, Tyrode-preserved, and quick-frozen lyophilized aortic segments, quick-frozen lyophilized grafts compare favorably with fresh and Tyrode-preserved segments. These segments apparently lie somewhere between fresh and Tyrode-preserved in stress-strain characteristics.

## COMMENTS

Today the responsibility of the surgeon caring for trauma cases lies not only in the recognition and treatment of bony, gastro-intestinal and parenchymatous damage, but also damage to the vascular system of either a primary or secondary nature. To accomplish this some knowledge of the available tools for arterial replacement is necessary.

The problem of arterial replacement in small vessels is easily dealt with by means of autogenous vein grafts. That this type of replacement has stood the test of time is substantiated by the reports of Murray,[10] Holden[11] and others.[12] Morphologically, this type of arterial replacement gives rise to a growing viable tissue which is not replaced by ingrowth from surrounding tissues as occurs in other types of arterial protheses.

The autogenous vein grafts fall short of the ideal in trauma or emergencies of the great arterial tree. It is here that an arterial prostheses must be used to correct the disorder. When Gross, Hurwitt, Bill and Pierce[13] reported their clinical use of the preserved aortic graft, the first clinical step had been taken. Carrel[14] and Guthrie[15] in the early part of this century had by experimentation founded the principle of arterial replacement and its possible clinical application. It is not in the functional results that this type of graft is limited, but in the methods of preservation. Preservation of these grafts is usually by means of a nutrient media which is refrigerated in a blood vessel bank. This type of preservation is effective for a thirty- to sixty-day period at which time the grafts, if not used, mature and must be discarded. The problems of a blood vessel bank are two in number. The principle one is that of procurement and to have the product mature and be discarded is so discouraging that the enthusiasm soon diminishes among the house officers and there is no material readily available when an emergency arises. The secondary problem is the customary six-hour deadline after the donor patient has expired. Experimentally (Table II) we investigated this question in a series of four animals with grafts which had been secured twenty-four hours after the donor dog's death and kept in Tyrode solution for seven days. One had a positive culture. (Table II.) Figure 5 shows the specimen obtained at death from one of these animals. Grossly the graft is adequate and functioning normally. It cannot be stated as yet,

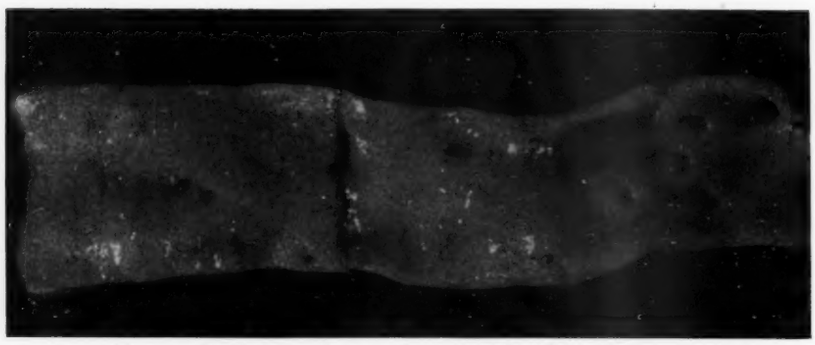


FIG. 5. Graft from dog 102 four months after placement.

but perhaps some of these grafts could be obtained at longer intervals than six hours from the time of death.

Experimentally we have found that with lyophilization some of the difficulties arising from vessels preserved in nutrient media are eliminated, since these grafts can be kept for protracted periods even at room temperature and thus loss of the grafts does not occur at a thirty- to sixty-day deadline. It also could lead to a central processing area for lyophilizing blood vessels to which several institutions could contribute their blood vessel segments and call upon the bank as their demands necessitated.

TABLE II

HOMOGENOUS AORTIC GRAFTS TWENTY-FOUR HOURS AFTER DEATH

| Dog | Operated | Culture + or − | Aortogram Date and Finding | Clinical Follow-up | Remarks |
|---|---|---|---|---|---|
| 101 | 2/26/52 | Not reported | Not done | 5/28/52, no femoral pulses | At postmortem on 3/28/52 specimen showed complete obstruction of lumen and scarring of graft |
| 102 | 2/28/52 | Bacillus subtilis | Not done | 4/19/52, dog sick, treated with penicillin; 7/2/52, dog died | At postmortem dog had peritonitis but graft patent and grossly functioning without difficulty |
| 103 | 3/18/52 | Not reported | 4/19/52, patent | 7/2/52, good femoral pulses | Three month follow-up with good functioning |
| 104 | 4/9/52 | Not reported | 4/23/52, patent | 7/2/52, good femoral pulses | Two and a half month follow-up with good functioning |

## CONCLUSIONS

1. Lyophilization of aortic homografts can be easily accomplished by simple available means.

2. The patency rate of such arterial grafts in dogs is high and has been proven by aortography, postmortem examination and clinical examination. The period of observation has been short (six months).

3. Histologically, these grafts are replaced by the host tissues.

4. "Quick-frozen" lyophilized grafts are superior to slow-frozen grafts, as in this method there is no disruption of cells due to the formation of macrocrystals.

5. Lyophilization of blood vessels could solve the disadvantage of maturing which is the greatest single deterrent in present day methods of blood vessel preservation and banking.

6. Three methods of arterial replacement are recommended in trauma: autogenous free vein grafts in the small arteries; preserved arterial homografts in the great arteries; and lyophilized arterial homografts in future blood vessel banking and great arterial replacement.

## REFERENCES

1. CARREL, A. Heterotransplantation of blood vessels preserved in cold storage. *J. Exper. Med.*, 9: 226, 1907.

2. Villard, E., Tavernier, L. and Perrin, E. Recherches experimentales sur las greffes vas aul ares. *Lyon cbir.*, 6: 149, 1911.
3. Yamanouchi, H. Über die zirkularen Gefabnahte und arterien venen-anastoznosen, sowie über Gefabtrausplantationen. *Deutscbr. f. Cbir.*, 112: 1, 1911.
4. Swan, H., Robertson, H. and Johnson, M. E. Arteria homografts. 1. The fate of preserved aortic grafts in the dog. *Surg., Gynec. & Obst.*, 910: 568, 1950.
5. Schloss, G. and Shumacker, H. B., Jr. Studies in vascular repair. IV. The use of free vascular transplants for bridging arterial defects. An historical review with particular reference to histological observations. *Yale J. Biol. & Med.*, 22: 273, 1950.
6. Harkins, H. N. Reports of National Inst. of Health Study Section. Unpublished data. Bethesda, Md., 1952.
7. Marrangoni, H. G. and Cecchini, G. P. Homotransplants of arterial segments preserved by the freeze-drying method. *Ann. Surg.*, 134: 977, 1951.
8. Carrel, A. Latent life of arteries. *J. Exper. Med.*, 12: 460, 1910.
9. Evans, F. G. and Lebow, H. M. Regional differences in some of the physical properties of the human femur. *J. Appl. Physiol.*, 3: 563, 1951.
10. Murray, G. Surgical repair of injuries to main arteries. *Ann. Surg.*, 83: 480, 1952.
11. Holden, W. Reconstruction of the femoral artery for arteriosclerotic thromboses. *Surgery*, 27: 417, 1950.
12. Johnson, J., Kerby, C., Grifenstein, F. E. and Castillo, A. The experimental and clinical use of vein grafts to replace defects of large arteries. *Surgery*, 26: 915–955, 1949.
13. Gross, R. E., Hurwitt, E. S., Bill, H. H. and Pierce, E. C. Preliminary observations on the use of human arterial grafts in the treatment of certain cardio-vascular disorders. *New England J. Med.*, 239: 578, 1948.
14. Carrel, A. Ultimate results of aortic transplantation. *J. Exp. Med.*, 15: 389, 1910.
15. Guthrie, C. C. Heterotransplantation of blood vessels. *Am. J. Physiol.*, 19: 482, 1907.

## DISCUSSION OF PAPERS BY GUY AND WICHOWSKI, AND JORDAN, HIERTONN AND JOHNSTON

Louis G. Herrmann (Cincinnati, Ohio): I should like to comment on one of the statements made by Dr. Jordan. I must disagree with him that "nothing can be done for extensive injuries to large arteries unless the surgeon has a piece of preserved artery available with which he can re-establish the continuity of the artery." I did not have a chance to read his paper before it was presented here, and I may have misunderstood the statement.

It has been our recommendation for many years that, in an emergency, the continuity of a large artery may be re-established temporarily by the use of any type tube. We have used glass tubes, Vitallium tubes and plastic tubes. Tuffier used silver tubes as long ago as 1917 and he showed that an extremity often can be saved by this emergency procedure. It is now well established that clotting of the blood will take place within the lumen of such tubes, but the acute ischemia of the extremity can be prevented and time gained for the development of a collateral arterial circulation.

Most surgeons who deal with the results of trauma are certain to be confronted by this problem at some time and it is our recommendation that Vitallium or plastic tubes be kept easily available for such an emergency. At a subsequent elective procedure replacement of this arterial segment can be done safely by the methods outlined by Dr. Jordan and his associates.

Edward A. Kitlowski (Baltimore, Md.): I was very much interested in Dr. Guy's presentation, and I want to stress the value of careful history taking and diagnosis in those cases. Three such cases have come into our hands, two of them winding up with more or less disastrous results. One of them was a young woman who developed a sharp pain on the inner side of her leg just above the popliteal space during delivery. Subsequently, in the next two or three weeks, a firm mass began to develop. A biopsy was taken of this area and a diagnosis of a round cell sarcoma was made. The patient was immediately given intensive x-ray treatments, with the idea that that would at least prolong her life to a certain extent. This resulted in very extensive x-ray destruction, and she came into our hands. We checked back on those microscopic sections, and pathologists confirmed our feeling that it was an organizing hematoma which had been mistaken for a sarcoma.

The second case was a girl who had been in an automobile accident and subsequently developed a long, cord-like tumor extending from the gluteal fold down through the popliteal space to the midcalf. This was biopsied and, again, a diagnosis of sarcoma was made. The patient was given innumerable treatments with radon seeds. Subsequently she lost her foot and had to have an amputation below the knee because of the destruction in the vascular and nervous systems.

In the third case a woman had a sharp pain while at work in an airplane factory. Subsequently a firm mass developed just below the head of the deltoid muscle. This grew larger slowly and then finally remained at a size which we have not been able to evaluate because we have not gotten all the data from the surgeon who had taken care of her, but the microscopic report again gave us to believe that it was an organizing hematoma with round cell infiltration which was again mistaken as some kind of malignant tumor. Extensive destruction was done of a portion of the deltoid.

In every one of these cases we believe that had a careful history been taken, and knowing that you do not get sarcomas like rods running up and down the leg or that they do not develop all of a sudden in cases in which there is a possibility of acute trauma, as in the case of the other two, probably more conservative treatment would have saved these people tremendous pain and discomfort and a great deal of functional loss.

SAM F. SEELEY (Washington, D. C.): With reference to vascular injuries I would like to add a progress report in keeping with the re-establishment of continuity of great vessels. You will recall that last year we reported on 106 cases of arterial aneurysm or arteriovenous fistula. At the present time we have had 151 such cases at Walter Reed, the vascular center for the Army and Air Force. After the extirpation of twenty-five fistulas which demonstrated vascular insufficiency of 29.6 per cent, we resolved that we would no longer extirpate. After that in sixty-seven consecutive cases re-establishment was attempted, with only two cases in which it could not be done; therefore there were sixty-five cases of re-establishment of continuity and only four failures.

There can be no doubt about the necessity of re-establishing vascular continuity. As Dr. Herrmann stated it might be done as an emergency.

The use of autogenous veins in our hands is entirely satisfactory, having used them in eleven cases. We have used homologous arteries in six cases.

We have now gone one step further. Our research team sent one of our young surgeons to Korea, where he tackled seventy-seven consecutive cases of vascular injury of the acute war wounded. Of these seventy-seven cases, forty-three were found to be of minor vessels which were ligated. In thirty-four cases, wounds involved major vessels, and re-establishment of continuity was successful in thirty-one cases. Some were as late as eighteen hours. I believe only four vein grafts were used, veins from the same individual. This demonstrates, then, that we can reduce the number of amputations by assignment of well trained young, steady-handed vascular surgeons to the forward hospitals.

ISIDORE COHN (New Orleans, La.): It is a pleasure for me to hear the progress which is being made. I would like to pay tribute to one to whom we owe so much historically. The pioneer in vascular surgery is none other than the master of American surgery, Rudolph Matas of New Orleans. The first experimental work on vascular surgery, as you recall, was done by Alexis Carrel, but prior to that, as early as 1888, Dr. Matas, encountering an axillary aneurysm, used a suture for the first time. I think all of the advances which have been made, particularly in arteriovenous fistula, to which General Seeley referred, we owe our earliest work to Rudolph Matas. At this time, particularly since he has just passed his ninety-second birthday, we should not overlook the pioneer to whom we owe so much.

LAURIE H. MCKIM (Montreal, Quebec, Can.): This question of the rupture of blood vessels is of special interest to me inasmuch as I have seen approximately six cases of rupture of the inferior epigastric artery. Two of these are reported in the literature which was reviewed by me about twelve or fifteen years ago. I would like to compliment Dr. Guy on his masterly presentation of the subject. However, the question is one that will bear a good deal of investigation. Dr. Kitlowski has mentioned the importance of history and of diagnosis. The history, however, may be misleading. The late Dr. Fraser Gurd, so well known to the older members of the society, had a very interesting case: A rather heavy, middle-aged man had a severe fall. He was brought to the hospital, and Dr. Gurd saw him and made a diagnosis of acute appendicitis. He was taken to the operating room and the ordinary transverse appendicular incision was made. Upon opening the rectus sheath it was found that it was filled with blood. The rectus muscle was partly torn and the inferior epigastric artery was bleeding rather profusely. This was controlled, but Dr. Gurd said, "This man has an acute appendix." He opened up the peritoneum, and the man had a moderately acute appendix.

This brings up, of course, the question of whether there is such a thing as trauma to the appendix, but still it bears on the subject that Dr. Kitlowski has mentioned, the importance of the history and the clinical examination. In other words, things are not always what they seem.

# RELATION OF TRAUMA TO LESIONS OF THE MALE BREAST*

W. Wallace Greene, M.D. and Nelson J. Howard, M.D.
*San Francisco, California*

EFFORTS have been made in the past to determine whether or not trauma plays a part in the production of hypertrophy of the male breast. The possibility has usually been considered when studies of gynecomastia have been made, but after briefly discussing such a possibility and stating the percentage of patients giving a history of trauma before the onset of the condition, the whole idea is usually dismissed because there is no proof and no control cases. In many papers on this subject the author has finally come to the conclusion that trauma has merely brought the condition to the patient's attention rather than that it was an etiologic factor. In view of these considerations and the fact that several cases were seen which seemed to be due to trauma, an effort was made to find cases which would tend to establish trauma as a definite causative factor. Cases seen in private practice and at the Stanford Clinic and the San Francisco Hospital were studied.

Much confusion exists in regard to the condition usually called "gynecomastia." However, as more and more studies have been made, it has become evident that such a term is merely a "catch-all" term and is not satisfactory. A better term is "hypertrophy of the male breast" and is the term now used by the Quarterly Cumulative Index Medicus. This change of terminology is desirable because it is now realized that many different conditions can cause a change in the form and volume of the male breast.

In addition to the confusion which surrounds the terminology, there is also considerable confusion as to the factors which exert an influence on the male breast as well as their mode of action. Hypertrophy occurs in men treated with estrogen, gonadotropins or methyl testosterone and often follows the treatment of Addison's disease with adrenal hormones and has been reported following the treatment of hyperthyroidism with radioactive iodine. The hormonal imbalance which undoubtedly plays a part in hypertrophy in many cases is difficult to determine.

Nutritional deficiency and chronic debilitating conditions, such as cirrhosis of the liver, are known to be associated with hypertrophy of the male breast. The occurrence in such cases seems to be best explained by the liver dysfunction which occurs with an inability of the liver to inactivate estrogens and a resultant accumulation in the blood. On the other hand, the "refeeding" hypertrophy observed in prisoners of war is difficult to explain and indicates that other factors such as certain amino acids may play a part in its production.

The occurrence of hypertrophy in cases of obvious endocrine dysfunction are not so hard to understand. Included in this category are those patients with atrophy of the testes which may follow trauma to or disease of the testes and also those cases of atrophy occurring in patients with conditions affecting the central nervous system such as those with injuries to the spinal cord.

Neoplasms of the testes may also result in hypertrophy. The most common of these are the teratomas although the condition is also found in those patients with chorio-epitheliomas, seminomas, adrenal tumors, hypophyseal adenomas, etc.

The occurrence in Klinefelter's syndrome further adds to the confusion. In these cases there is no apparent testicular deficiency and it seems that there may, in fact, be an increased amount of androgen present. This may be in line with the hypertrophy produced by the administration of testosterone in hypogonad patients and has led some to postulate some other testicular hormone which inhibits androgenic activity.

This means that in any case having hypertrophy of the breast, all of these possibilities must be kept in mind. All such patients must have a careful and complete history and physi-

* From the Department of Surgery, Stanford University Medical School, San Francisco, Calif.

cal examination. This becomes particularly important when one bears in mind the possibility that the hypertrophy may be only the effect of some serious underlying condition.

Unilateral hypertrophy is difficult to explain unless one considers other factors which might make one breast more susceptible to hormonal influences by lowering the threshold of the target area to varying amounts of hormones or changes in the estrogen-androgen balance. It would seem only reasonable if some hormone factor was at work that it would exert the same influence on both breasts with resultant bilateral hypertrophy although it is possible that over a period of time the second breast might become involved at a later date. However, the bilateral cases make up only about 10 to 15 per cent of the total in most series and this would seem to allow for the consideration of factors which might make one breast more susceptible than the other to the various factors acting on the breast. We believe that trauma is one of these factors. In nearly all series reported, trauma is not noted in the histories of those cases in which there were definite signs of endocrine dysfunction.

The literature contains many references to the effect of trauma on the breast and there have always been enough histories given by patients that seemed authentic so that at least in some it would seem that trauma played a part. One of the earliest references in American literature was a case reported by Peters in 1863 in the American Medical Times. The report was one of a soldier who three years before being seen had been caught between the wall of a building and some machinery and received some contusions to his left shoulder and the left side of his chest. He subsequently developed hypertrophy and pendulousness of the breast on that side. Deaver and McFarland found seven cases up to 1917 which gave histories of trauma prior to the hypertrophy "without a suspicion of heredity, degeneration, or sexual defect." Their conclusion was that hypertrophy "may follow local injury to the breast" and they chose to call the condition "chronic indurative mastitis." They stated that they were not sure that such a condition was caused by the same process which caused other forms of gynecomastia and stated further that once the enlargement occurred it was permanent.

Several series have been reported (Geschickter, Karsner, Lenson, Menville, Webster) in which a history of trauma was given in from 10 to 22 per cent with the breast changes beginning in from a few hours to several years after the history of injury. In a series of forty-six cases previously reported by one of us (W. W. G.) a history of trauma was obtained in 17 per cent. In those with a long history between the time of injury and the onset of the hypertrophy, it is more difficult to prove a causal relationship than in those in which the hypertrophy began within a comparatively short time after injury. In all series reviewed the authors stated that the relationship could not be proved in their cases. Karsner stated that trauma or irritation may draw attention to the lesion and may augment growth but that there is no satisfactory evidence that either one is causative. Menville found a history of trauma in 14 per cent of 130 cases. He concluded that "it is questionable whether trauma plays any particular role in this phase of activity, although it is probably more closely associated with unilateral growth." Webster stated that the injuries were probably sustained by an enlarged breast and that the injury probably resulted in exaggeration of the deformity with resultant pain and disability. Such statements influenced us to review this subject and see whether or not some evidence could be found to the contrary.

Included in this paper are some data on twenty-one patients with hypertrophy of the breast who gave histories of injury followed by hypertrophy. (Table 1.) Some of these cases were included in an earlier study (W. W. G.) but several other cases have been seen and they have been added to the group and a new study made. The ages of the patients ranged from fifteen to seventy years of age. The average was forty-two years. The time interval between the history of injury and the onset of hypertrophy varied from one week to one and one-half years. It occurred in the left breast in eight cases, in the right breast in eleven cases and in both in two cases. The types of injury were many and varied. In eight of them there was a history of a single injury to the breast such as being struck with a baseball, a swinging gate, the handle of a shovel, a blow with a fist, pinching, etc. The trauma was repeated or was present for a considerable period of time before a mass appeared in the breast in the other cases.

In our opinion, it seems justifiable to state that trauma is a factor in the production of

hypertrophy if an injury occurs to a previously normal breast, if the trauma was severe for a short time or of lesser degree for a longer time so as to give a definite history of trauma, and if the patient had persistent symptoms from the time of the injury to the time of appearance of the hypertrophy.

In nearly all of these cases there were persistent symptoms from the time of injury to the time of appearance of the hypertrophy, although in several of the cases there was no statement made as to whether or not the symptoms were continuous. The most interesting cases in the group are the last six cases shown in the case summaries. These men were all examined prior to employment and were passed as being in good physical condition. They all did the same kind of work and were all right-handed. During the course of their work they rubbed the right sides of their chests against the top of a large centrifuge while leaning over to clean it. After varying time intervals they all developed hypertrophy of the right breast. Three had simple mastectomies and two of them were diagnosed by the pathologist as "gynecomastia" while the third was told his tumor was "OK." Two others were changed to other jobs so that there was no further irritation of their breasts but they received no treatment. The mass disappeared from one in six months and from the other in four months without any recurrence. The third one which was not operated left the plant one month after being changed to another job and efforts to contact him were unsuccessful so there was no follow-up on him. These cases certainly indicate that trauma was a factor in producing the hypertrophy. There is further evidence in the fact that whereas six cases occurred in the space of a year in this plant in men on this job, no more cases were seen after some changes were made in the construction of the centrifuge and the employees were given a platform to stand on so that their chests did not rub on the edge of the machine.

As stated previously, it is difficult to determine the mechanism of the hypertrophy and why it occurs. It would seem that trauma acts in making the target organ more susceptible to the various stimuli which act on the breast.

Various forms of sensory stimulation have their effect on endocrine end organs. A pseudopregnant condition may be induced in the female rat by sterile copulation or cervical stimulation. Again virgin adult female rats may be made to suckle newborn litters. The dead white small nipples of the virgin female rats become elongated, swollen and pink within three days with enlarged pink areolae and on continued suckling, colostrum is produced. Microscopically, one can show in such adult rats the growth and development of the alveolar secreting tree from the simple branching tubes and ducts of the adult virgin female.

The nipple, areola and breast tissue are erectile tissue with definite vascular response to sensory stimulation. Trauma, either single and severe with the subsequent inflammatory reaction to tissue damage, or more frequently repeated minor trauma or continued sensory stimulation evoke the maximum vascular engorgement. When one breast is constantly bathed in an increased and augmented blood supply, the estrogenic substance or steroid hormones normally present in the blood stream may be brought to the breast in quantities beyond the threshold level and unilateral hypertrophy of breast tissue takes place.

Perhaps the mechanism is similar to that seen recently in a nine year old girl. Her right breast was pinched hard by a playmate at school and the breast remained tender from that time on. After three or four weeks the breast began to increase in size and remained quite tender. It developed a 2 cm. rubbery button under the nipple. This has remained with little further change to date. This child's breast tissue was undoubtedly similar to that of the normal male breast. At her age with no secondary sexual characteristics present, it would seem that the trauma altered the breast tissue in some manner so as to make it more susceptible to some growth-producing influence.

The pathologist's findings do not give much aid in determining the factors producing hypertrophy. Many different terms are used by the pathologists in describing the hypertrophy found in so-called gynecomastia. The reason for this probably is that the appearance of the tissue differs in some respects in each individual case. However, they are probably all different stages of the same condition. In general, the findings may be almost identical with those of the normal pubescent female breast. There is usually only moderate hypertrophy of the gland elements with usually no increase in fatty tissue. The periductal connective tissue is increased and the ducts are usually dilated and

TABLE 1
DATA ON TWENTY-ONE PATIENTS

| Patient | Age | History | Physical Examination | Follow-up | Pathologic Report |
|---|---|---|---|---|---|
| E. J. (Stan.) | 57 | Wife bumped his breast with elbow | 2½ × 2 cm. smooth non-tender mass (rt.) | Excised; nipple saved | Benign fibro-adenoma |
| R. R. (Stan.) | 43 | Struck with baseball 15 yr. before; immediate swelling which subsided, breast remained larger than other; rapidly increased 1 yr. before being seen | 1 cm. non-tender mass, like small female breast (lt.) | Excised; nipple saved | Gynecomastia |
| W. F. (stan.) | 28 | Repeated injuries left breast while playing football; mass appeared 1 yr. before and increased in size, then decreased to form smaller nodule | 3.5 cm. sl. irreg., sl. tender (lt.) | Excised; nipple saved | Gynecomastia, mastitis |
| H. A. (Stan.) | 65 | Struck by swinging gate, remained tender; 1 mo. later mass appeared | 1½ cm. nodule, sl. tender (lt.) | Excised nipple | Hyperplasia of male breast (gynecomastia) |
| A. S. (Stan.) | 22 | Struck with fist | Smooth rubbery mass 1.5 cm.; not tender (lt.) | Excision advised, but refused; returned 4 mo. later and mass was gone | |
| A. N. (Stan.) | 15 | Struck 7 yr. before; began to enlarge in 1 yr.; stayed present size 5½ yr. | Ropy tissue like cystic mastitis; not tender (lt.) | Biopsy advised; did not return | |
| L. W. (S.F.H.) | 61 | Carried heavy cash box against chest while at work and noticed lump in breast 2 mo. before; had been seen 3 yr. before with mass in rt. breast, no definite history of injury; tender 6 mo. before mass | 2–3 cm. mass under nipple; not tender (lt.) | Simple mastectomy | Chronic mastitis |
| | | | Small firm mass not tender (rt.) | Simple mastectomy | Adenofibroma |
| S. S. (S.F.H.) | 49 | Bruised nipple with coil of rope; 5 days later noticed sl. discharge from nipple; lump continued to increase in size for 6 weeks. | 4 cm. indurated mass; sl. tender (rt.) | Simple mastectomy | Chronic mastitis with abscess formation |
| W. N. (S.F.H.) | 37 | In body cast after laminectomy; lay on left side most of time because of pain; after 4 weeks left breast tender; 2 weeks later mass appeared | 1½ cm. mass; sl. tender (lt.) | Excised; nipple saved | Fibro-adenomatous hyperplasia; chronic mastitis |
| C. D. (S.F.H.) | 65 | Used crutches after "stroke"; rt. breast became tender and swollen where crutch rubbed against chest | Sl. tender (walnut sized nodule) (rt.) | Simple mastectomy | Cystadenoma breast |

TABLE 1 (*Continued*)

| Patient | Age | History | Physical Examination | Follow-up | Pathologic Report |
|---|---|---|---|---|---|
| J. M. D. (E.J.W.) | 50 | In body cast after laminectomy, 1 wk. later rt. nipple tender, enlarged where edge of cast rubbed; cast removed 4 mo. later and mass present | Tender 2 cm. mass (rt.) | Simple mastectomy | Chronic cystic mastitis |
| W. J. (W.W.G.) | 41 | Suspenders rubbed over breast and caused irritation; after few weeks lumps appeared in breasts | 2 cm. mass both breasts; not tender (both) | Observed; advised wear belt; after 3 mo. lesions only half as large; compl. gone 5 mo; no recurrence | |
| F.M.R. (W.W.G.) | 33 | In body cast for back and hip injury; top of cast irritated left nipple; day after cast applied; remained tender; 6 wk. later small mass appeared under nipple | 2 cm. mass; sl. tender (lt.) | Excised; nipple saved | Gynecomastia; hyperplasia with fibrosis |
| D. K. (W.W.G.) | 43 | Right breast pinched severely by another man while scuffling; breast tender 6–9 mo. and then mass appeared | Tender mass about 2 cm. in diam. under right nipple | Simple mastectomy; healed with large scar | Benign hypertrophy (patient told it was all right) |
| E. C. (W.W.G.) | 70 | Struck breast with handle of shovel; remained tender and noticed lump; time interval 4 wk | 2½ cm. mass; sl. tender (lt.) | 10 mg. testosterone daily for 2 wk.; mass began to subside by 3rd wk. and gone in 6 wk.; no recurrence | |
| P. O. (J.P.) | 30 | Irritated rt. breast by rubbing chest repeatedly against edge of large centrifuge while cleaning it; mass appeared after 2 mo. | Tender small mass (rt.) | Simple mastectomy | Gynecomastia |
| R. C. (J.P.) | 23 | Same history as above; mass after 1 mo. | Tender enlargement (rt.) | Mass still present 1 mo. after changing jobs; he left the plant then and was not seen again; follow-up cards not answered | |
| E. K. (J.P.) | 28 | Same history as above; mass after several months | Mass size of walnut; tender (rt.) | Changed jobs; slightly smaller in 3 mo. and disappeared in 6 mo.; no recurrence | |
| A. P. (J.P.) | 33 | Same history as above; mass after 18 mo. | Enlarged, painful (rt.) | Simple mastectomy | Patient told was "O.K." |
| W. G. (J.P.) | 27 | Same history as above; mass after 1 yr. | Enlarged, painful (rt.) | Simple mastectomy | Gynecomastia |
| D. I. (Ins.) | | Same history as above; mass after several mo. | Small mass, tender (rt.) | Disappeared in 3–4 mo. after changing jobs | |

are even increased in many cases. In a few there is actual branching of the ducts but, as a rule, no true acini are present. Epithelial hyperplasia of the linings of the ducts is present and this is often thrown up into papillary projections. In some cases there is desquamation of the cells into the lumen and occasionally secretion is present in the ducts. Lymphocytic and plasma cell infiltration with increased numbers of eosinophils is frequently found. Occasionally a fair number of polymorphonuclear cells are present. In most cases there is no evidence of encapsulation but this occurs in cases often labelled as "fibroadenoma." Probably this is just part of or an accentuation of the same process found in ordinary cases of hypertrophy. One possible explanation is that it is the result of hypertrophy of long duration. There seems to be no consistent difference in those with pain as compared with those without pain although some authors report more lymphocytic and polymorphonuclear cells about the ducts in those with pain.

In all cases of hypertrophy of the breast a definite effort must be made to determine the etiology of the condition. It is essential that one rule out the presence of neoplasm of the testes. Any mass in the male breast for which there is no definite diagnosis or in which the etiology is obscure should be removed just as in the female. Nodular masses and especially those which are hard must be removed. From the cases reported in this study it is evident that some of the cases of hypertrophy will subside without treatment. However, if the surgeon elects to observe such cases for a time, he must acquaint the patient with the possibilities and impress upon him the importance of regular and frequent observations. Some advocate the use of testosterone but there is no definite evidence to lead one to believe that this is advantageous especially since in most cases reported there has been a recurrence after the testosterone was discontinued.

These masses in the male breast are easily removed with a semi-circular incision at the margin of the areola. In this way the nipple can be saved. Care should be taken to preserve the blood supply to the areolar and nipple area. Accumulations of blood or serum under the areola can be prevented with careful hemostasis or the use of a small drain if necessary. The tissue removed must be carefully examined by a pathologist and, of course, if the tumor is malignant a radical breast amputation must be done.

## SUMMARY

1. Twenty-one cases of hypertrophy of the male breast are reported in which trauma appeared to be the etiologic factor. The trauma probably acted by changing the target area (breast) so as to make it more susceptible to hormonal influences.

2. Masses in the male breast must be removed for biopsy if there is any uncertainty as to their nature or origin.

*Acknowledgment:* We wish to thank Dr. E. J. Wylie and Dr. Joseph Pace for permission to include in this paper cases seen by them.

## REFERENCES

ALLEN, E., DANSFORTH, C. H. and DOISY, E. A. Sex and Internal Secretions, 2nd ed. Baltimore, 1939. Williams & Wilkins.

BAUER, F. K. and GOODWIN, W. E. Acne and gynecomastia following therapy for hyperthyroidism, *J. Clin. Endocrinol.*, 11: 1574–1576, 1951.

COOPER, I. S. and HOEN, T. I. Gynecomastia in paraplegic males (7 cases). *J. Clin. Endocrinol.*, 9: 457–461, 1949.

DEAVER, J. B. and MCFARLAND, J. The Breast: Its Anomalies, Its Diseases, and Their Treatment, Philadelphia, 1917. Blakiston's Son & Co.

GESCHICKTER, C. F. Diseases of the Breast. Philadelphia, 1945. J. B. Lippincott Co.

GREENE, W. W. Surgical conditions of the male breast. Read before the Pacific Coast Surgical Association, Feb. 28, 1952.

KARSNER, H. T. Gynecomastia. *Am. J. M. Sc.*, 214: 121–127, 1947.

KLEINFELTER, H. F., REIFENSTEIN, E. C., JR. and ALBRIGHT, F. Syndrome characterized by gynecomastia, aspermatogensis, without A-Leydigism and increased excretion of follicle-stimulating hormone. *J. Clin. Endrocrinol.*, 2: 615–627, 1942.

LENSON, N. Enlargement of the male breast in naval personnel. *Am. J. Surg.*, 82: 325–327, 1951.

MCCULLAGH, E. P. and ROSMILLER, H. R. Methyl testosterone androgenic effects and the production of gynecomastia and oligospermia. *J. Clin. Endocrinol.*, 1: 496–502, 1941.

MENVILLE, J. G. Gynecomastia. *Arch. Surg.*, 26: 1054–1083, 1933.

NEAL, M. P. and SIMPSON, B. T. Disease of the male breast. *J. Missouri M. A.*, 27: 565–570, 1930.

PETERS, D. C. Hypertrophy of the mammary gland in a soldier. *Am. M. Times*, 6: 196, 1863.

SALTER, W. T., KLATSKIN, G. and HUMM, F. D. Gynecomastia due to malnutrition (prisoners of war). *Tr. A. Am. Physicians*, 213: 31–36, 1947.

SCHNURBUSCH, F. Untersuchungen über die Morphologie der mannlichen Brustdruse wahrend des

Lebensablaufes als Grundlage fur ein Studium der Gynakomastie, *Ztschr. f. Path.*, 62: 373–401, 1951.
WEBSTER, G. V. Gynecomastia in the navy. *Mil. Surgeon*, 95: 375–379, 1944.
WERNICKE, H. O. Gynecomastia. *Surgery*, 5: 217–225, 1939.

DISCUSSION

R. ARNOLD GRISWOLD (Louisville, Ky.): I would like to draw attention to the fact that in paratroop training centers a considerable number of cases of traumatic mastitis were observed due to slapping of the parachute harness on the breast during parachute drops.

H. MINOR NICHOLS (Portland, Ore.): I had one interesting case, a man about sixty-five years of age, who came in after he had developed bilateral swelling of his breasts. We went over him very carefully and could not find any reason for it at all. Finally, we found out that some other doctor had given his wife stilbestrol and the patient thought that if it were good for her, it was good for him. So he had been taking his wife's stilbestrol and developed a bilateral hypertrophy.

BYRON E. BOYER (Cincinnati, Ohio): This summer I had one case, a young man twenty years of age, who six years before began to develop bilateral hypertrophy of the breast. As a matter of fact, they looked like virginal breasts in the female. He was about six feet tall, weighed 190 pounds, had been married for two years but had no children. The internist could find nothing abnormal. In this particular instance I did a bilateral mammoplasty, using the Thorek procedure in transplanting the nipples.

# SHOTGUN WOUNDS*

JAMES C. DRYE, M.D. AND GEORGE SCHUSTER, M.D.
*Louisville, Kentucky*

IN recent years due to the impetus of widespread and continuous war, a great deal has been written about wounds produced by such missiles as bullets, shell fragments, bomb fragments and mines. None of these missiles produce the type of wound that is created by the load from a shotgun. Shotgun wounds, and they are fairly common, have been largely neglected in the literature. Other than a few reports of single cases and brief mention in papers mainly devoted to other types of wounds,[1–12] we have found no paper on shotgun wounds in American literature since that of Byrd Charles Willis published in 1935.[13] Willis's patients were all treated before the first of the sulfonamides and before the use of massive transfusions.

Statistics as to the incidence and mortality of shotgun wounds are almost meaningless because it is impossible to avoid a selection of cases which differs in each series. This unavoidable selection of cases is due to the extreme variation of the severity of injuries produced by the shotgun. The injuries vary from penetration of the skin by two or three pellets to a highly fatal, destructive, massive wound. However, these wounds are certainly common enough to be of definite interest, and the mortality is high when the patient is hit at close range and has received a large proportion of the load in a direct hit.

The type of wound produced is basically determined by the amount of scatter of the pellets. The severity of the wound is determined by the number and velocity of the pellets, and by the size when unusually large shot, such as 00 shot, are used.

These factors of scatter, number, velocity and size vary with different guns and with different types of load. Since it would be impractical to discuss all the variations of the different guns and different loads and since, from a practical standpoint, there is not too much difference, we will discuss these factors as they exist in one common type of gun and load, namely, the twelve-gauge gun firing the standard load of one-quarter ounces of number six shot.

If the target is in motion, another factor governing the number of pellets that hit the target comes into play. The pellets string out along the axis of the line of flight as well as dispersing perpendicularly to this axis. (Fig. 1.) Thus the pellets do not all hit the target at the same time. This shot string is about 6 feet long at 20 yard range and at 40 yard range, it is from 10 to 15 feet long. The target may thus move through the string and be hit with only the head or tail of it.

In passing, it is interesting to note how the gauge of shotguns was originally determined. After the barrel was made, a single solid shot was made of such size that it was just able to enter the muzzle of the gun. The number of such round shot that was required to make a pound gave the gauge. For example, if it took twelve such round solid shot to make a pound, the gun was a twelve-gauge gun.

The muzzle velocity of the shotgun is about 850 feet per second, which is about the same as that of a pistol and about one-third that of an Army Rifle. (Fig. 2.) The velocity of a shotgun falls off much more rapidly than does that of the pistol or rifle.

The twelve-gauge gun loaded with number six shot discharges 275 pellets. At a distance of 40 yards, a little over 200 of these will hit in a circle 30 inches in diameter. On a man about 6 feet tall and weighing 160 pounds, this circle would reach from approximately mid-thigh to the shoulders, completely across the trunk, and extend out 9 inches on each side. (Fig. 3.) Calculating the area exposed, about one-half of the shot, or 100, would hit the patient. If the shot were absolutely evenly distributed, they would be about 2 inches apart. This represents the maximum number of penetrations that could occur at this range. However, inasmuch as few people are likely to get hit with the center of the pattern of shot being centered directly

* From the Department of Surgery, University of Louisville School of Medicine, Louisville, Ky.

on the geometric center of the body, it is usual that fewer shot hit the patient.

We have had no experience with wounds produced by the larger shot. The diameter of the 00 approaches that of the bullet fired by the German Luger and will go clear through a man up to 40 yards or so. The twelve-gauge cartridge of 00 shot contains eight to ten of these pellets. Close range wounds produced by this load rarely reach the hospital. Scatter injuries produce the same type of wound as that produced by multiple small caliber pistol bullets and should be managed in the same way.

At ranges between 15 to 40 yards the twelve-gauge gun, loaded with number six shot, will produce small but deeply penetrating wounds, the number of which depends on how much of the pattern hits the patient, and varies from three or four to as many as a hundred or more.

Obviously, no standard form of therapy can be laid down for scatter type injuries inasmuch as they vary so widely. The patient should be carefully examined with particular reference to injuries of nerves, joints and special sense organs; and if such injury has occurred, appropriate treatment should be instituted. We had one patient who was struck by only three pellets, one of which severed the right median nerve. This was successfully sutured primarily.

When the abdomen is involved in this scatter type wound, there are a great number of visceral perforations, fifty to one hundred fifty or so. These perforations, in almost all instances, seal over. We have not seen personally, or found in the literature, a case reported in which the bowel was struck tangentially

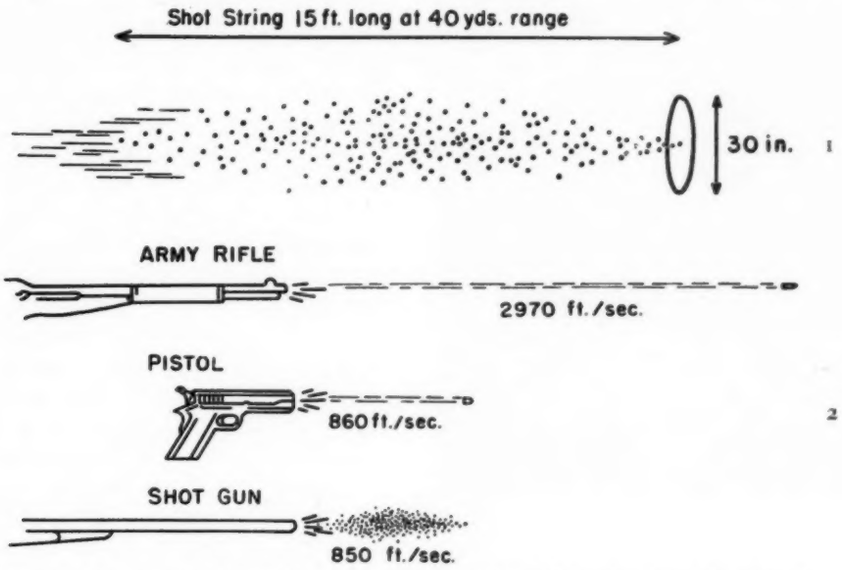



FIG. 1. Diagram of shot string at forty yards. The pellets are along the axis of the line of flight as well as perpendicular to it.

FIG. 2. Diagram showing relative velocity of an army rifle, pistol and shotgun.

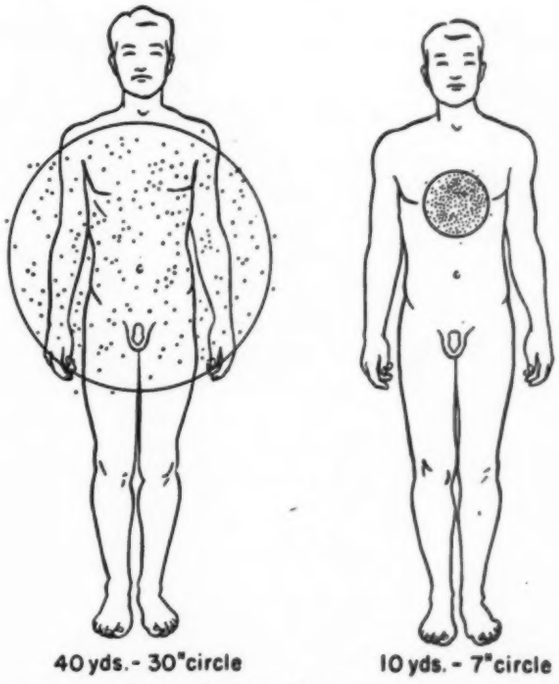



FIG. 3. Sketches showing the pattern of impact at 40 yards and at 10 yards.

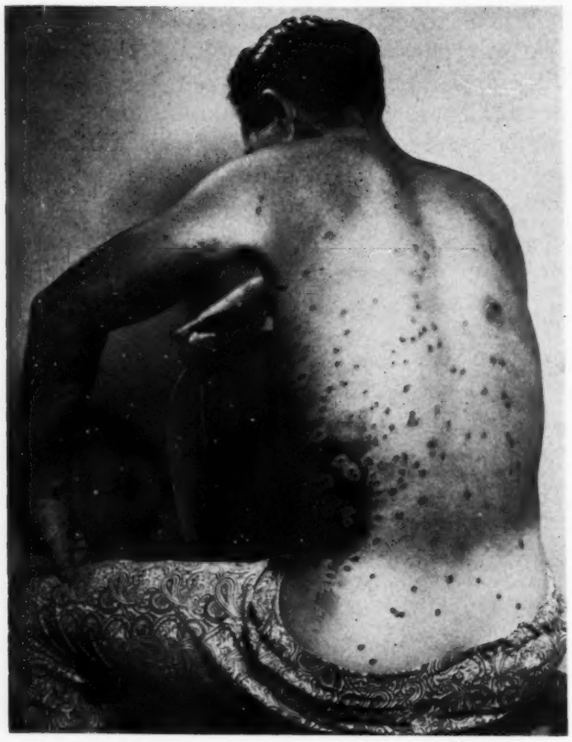


FIG. 4. Scatter type wound; range about 20 yards. There are 176 penetrating wounds.

and laid open in this scatter type of wound. If exploratory laparotomy is done in such cases, it is almost impossible not to overlook several of the perforations; and such an exploration would require the retroperitoneal exploration of the colon and duodenum. Handling the intestine leads to unavoidable soiling by intestinal contents squeezed out through the multiple bowel perforations into the peritoneal cavity. For these reasons we explore such cases only for definite indications such as delayed localized abscess. These factors were brought out by Willis in 1935, and we believe his reasoning is even more applicable today than it was at that time, inasmuch as the patient is protected from infection by the antibiotics.

I would now like to present to you one of two almost identical patients who happened to be admitted within a week of each other. This is a good example from our series of conservatively treated scatter type wounds of the abdomen. None of the patients of this series died or developed serious complications, or complications that would likely have been prevented by operation.

A twenty-six year old man was shot in the back at a distance of about 20 yards. (Fig. 4.) The gauge of the gun and the type of load are not known. On admission he was found to be in moderately severe shock and to be coughing up blood-stained mucus. He was found to have a 30 per cent pneumothorax on the left. The examination of the abdomen showed generalized tenderness, particularly on the left. No peristalsis was present. As can be seen in the illustration, the shot pattern hit mostly on his left side and extended from his shoulders to his buttocks. There were 176 wounds of entry. X-ray studies showed many shot distributed throughout the left lung field and inside the peritoneal cavity. The chest was aspirated and 450 cc. of blood were withdrawn. He was put on continuous gastric suction, with nothing by mouth, and parenteral fluids and antibiotics. Within three days the patient was able to get up, get around and was feeling well. He was discharged at the end of two weeks, after having shown signs of moderately severe peritonitis during the first three or four days.

Chest injuries produced by widely scattered, deeply penetrating pellets have all been treated expectantly. Thoracotomy has not been necessary. Collections of blood or air have been treated by simple aspiration.

At close range the gun produces an entirely different type of wound. At 10 yards, 200 pellets hit in an area 7 inches in diameter. (Fig. 3.) At still closer range the charge hits as an aggregate mass instead of as individual pellets. Under either of these two conditions all tissue in the path of the charge is either completely blown away, or so thoroughly shredded and contused as to be completely devitalized. Added to this effect there is a good bit of blast at extreme close range, and frequently the wadding of the cartridge and other foreign material, such as clothing, is carried into the wound. If the charge hits the body or extremities tangentially, there is an avulsion of all tissue, including nerves, blood vessels and even bone.

It has long been held that when a full load, fired from close range, penetrated the abdomen or chest, operation was useless. However, since massive blood transfusions and antibiotics have become available, this is no longer true. In the last five years, we have had seven such abdominal wounds. One was moribund on admission and died a few minutes later. The other six were operated upon; two lived and four

died. One died on the table and the other three within six hours of operation. None of the four who died had injuries which were necessarily fatal *per se*, but they died of hemorrhage and shock. Theoretically, we should have been able to save all six of them.

The first of the two successful cases was a twenty year old woman shot with a four- to ten-gauge gun held against the right lower quadrant of her abdomen. There was a one-inch round, ragged wound of entrance. After thirty minutes of an intensive antishock therapy and preparation, the abdomen was opened through a perimedian incision. It was found that the main charge had been delivered to the right lumbar gutter superior to the iliac arteries, inferior to the kidney and lateral to the spine. The right colon was almost unrecognizable as a structure; 2 feet of ileum had been blown away and there were three other transections of the small bowel. The roots of the right second, third and fourth lumbar nerves were destroyed. The wadding and some clothing were discovered in the right iliac fossa. A right hemicolectomy, with a primary end-to-end ileotransverse colostomy, was done. The 2 feet of small bowel were resected and continuity of this and the three other transections was re-established by primary end-to-end anastomosis. The wound of entrance was widely débrided. The retroperitoneal area was drained through a stab wound of the right flank. The incision was then closed with through-and-through sutures. After an only moderately stormy course, the patient was discharged thirty days later, and was back at work within three months.

The second surviving case was that of a thirty-six year old man who, while hunting, attempted to break up a fight between his dogs and a farmer's pig by poking at them with the butt of his twelve-gauge shotgun, loaded with number four shot. On admission there was a gaping 4 cm. wound at the left of the umbilicus and several loops of small bowel were eviscerated. After thirty minutes of intensive antishock therapy he was opened through a transverse incision, the left half of which incorporated the wound of entry. Beneath the skin it was found that the left abdominal muscles and fascia were destroyed from the border of the left rectus to the posterior axillary line, and from the rib margins above to well down below the unbilicus. There were

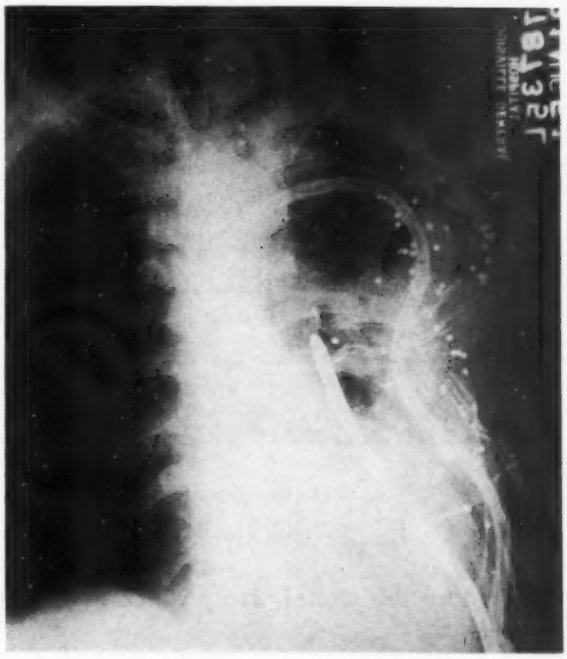
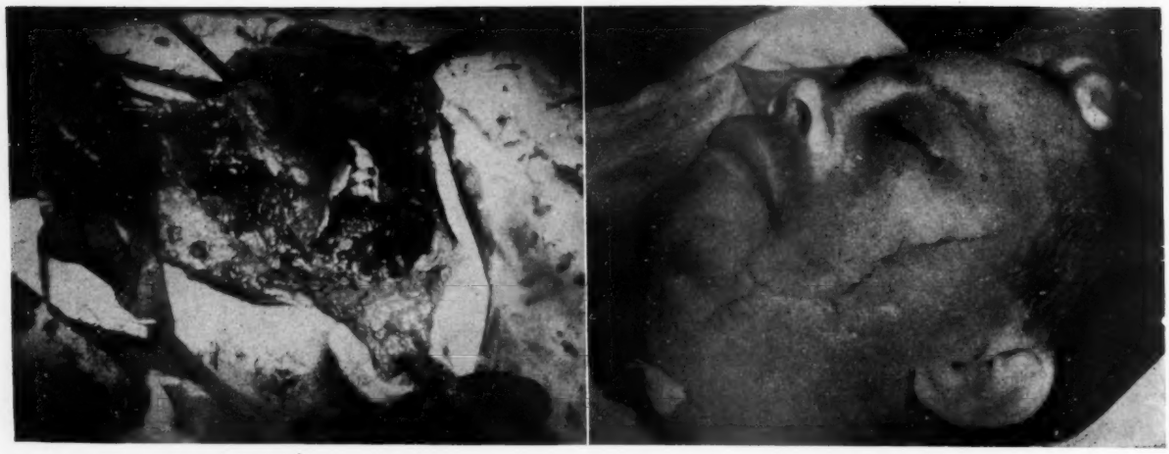


FIG. 5. Destruction of chest wall resulting from charge fired with muzzle held against skin of left chest.

two transections of the jejunum. There was another segment of jejunum, 35 cm. in length, severely damaged by several lacerations and perforations. There were a half dozen non-leaking penetrations of the sigmoid colon which were closed by suture. Appropriate resections and primary anastomoses were done. The biggest job was thorough débridement of the abdominal wall. The damaged abdominal muscles and fascia were removed which left a considerable defect on the left of the abdomen. This wound was partially closed with deep interrupted wire sutures, and the remaining defect was closed only with skin and subcutaneous tissue which was mobilized off the chest and lower abdomen. The wound was drained out through the left flank. The man ran a surprisingly uneventful course and was discharged on the thirtieth day. He is now back at work as a farmer. Nine months after injury a 7 cm. fascial defect is noted in the left abdominal wall but no real herniation. He wears an abdominal binder.

We have had two very short range massive injuries of the chest. One of these patients, shot at a distance of 10 feet, had the upper lobe of the left lung severely damaged and there was extreme damage of the chest wall. However, his postoperative death was due to hemorrhage



6 7

FIG. 6. Destruction of face resulting from charge fired with muzzle held under chin.

FIG. 7. Result obtained in patient shown in Figure 6.

from a torn intercostal artery which had been overlooked.

The second, a suicide attempt, held the muzzle of a twenty gauge shotgun against his left chest and shot himself. (Fig. 5.) There was a sucking wound 3 inches in diameter in the chest wall. The third to seventh ribs on the left were shattered and the lung was badly lacerated. There were five perforations into the heart. The lung tissue was débrided and closed. It was not necessary to do a decompression of the pericardium. The chest wall was thoroughly débrided and an air-tight closure was obtained by the use of flaps of skin and subcutaneous tissue. The pectoralis major muscle had been largely blown away. The patient survived but developed a postoperative empyema. Several months later this empyema was found to be of tuberculous origin and a successful pneumonectomy has recently been done. At present, the patient is progressing nicely but has not as yet gone back to work.

Shotgun wounds of the head are almost always serious. In six cases we had two deaths, and there were serious complications in the other four patients. One patient died from meningitis and one from massive brain damage. One patient lost an eye, one had motor aphasia, one developed epilepsy and one has a permanent seventh nerve paralysis.

Short range direct hits in the head are always fatal. Close range tangential wounds produce a varying loss of tissue. Débridement need not be as radical as in other parts of the body, but it must be thorough. However, in many cases more tissue can be saved than appears on the first inspection of the wound.

A thirty year old white male placed the muzzle of a two-ten guage shotgun under his chin and shot himself. (Fig. 6.) On admission the patient was in profound shock and after antishock therapy he was taken to the operating room. It was found that the main part of the charge had gone superiorly and laterally to the left. It had involved the floor of the mouth, part of the tongue, the mandible, zygoma and maxilla. The latter bones were badly fragmented. After extensive débridement, the mandible was fixed with internal wire fixation, the maxilla by an internal splint and the soft tissues were closed tight. (Fig. 7.) Other than the development of a panophthalmitis due to the penetration by one pellet, which required an evisceration of the left eye, the patient made an uneventful recovery. He was left with a left seventh nerve paralysis.

In closing, there are certain features of shotgun wounds and principles of management of these wounds that we would like to emphasize. At short range the shotgun produces a wound in which some tissue is completely blown away and much is badly damaged by shredding and by contusion. The débridement must be thorough if infection is to be avoided.

Further, some foreign material is almost always carried into the wound. This frequently includes the packing from the cartridge. All foreign material must be removed and a particularly careful search made for the packing. This packing, if left in the wound, is a frequent

source of tetanus and always leads to abscess and/or fistula formation.

Primary closure by use of undermined skin flaps or skin grafts should be done. Closure will help prevent infection and certainly prevents large losses of protein and fluids from these large wounds. Healing and recovery are much more rapid.

Following these principles, we have not had any patient develop tetanus or gas gangrene in our series of over fifty patients treated in the last five years, and our wound healing has been satisfactory for this type of wound.

REFERENCES


1. Anderson, H. M., Harris, W. G. and Bowen, F. H. Management of perforating and penetrating wounds of the abdomen. *J. Florida M. A.*, 34: 590–592, 1947–1948.
2. Binkley, R. W. and Cole, E. R. Gunshot wounds of the abdomen. *California & West. Med.*, 45: 26–30, 1936.
3. Black, B. M. Surgical treatment with recovery in a case of perineo-abdominal shotgun wounds from close range with multiple injuries to viscera. *S. Clin. North America*, 24: 952–958, 1944.
4. Bolton, P. Multiple birdshot wounds. *Ann. Surg.*, 34: 419, 1901.
5. Grace, K. D. Gunshot wounds of the abdomen. *South. Surgeon*, 14: 202–208, 1948.
6. Oberhelman, H. A. and LeCount, E. R. Peace time bullet wounds of the abdomen. *Arch. Surg.*, 32: 373–412, 1936.
7. Ritter, H. H. Penetrating wounds of the abdomen inflicted by firearms. *West Virginia M. J.*, 36: 107–112, 1940.
8. Rippy, E. L. Perforating gunshot wounds of the abdomen. *J. A. M. A.*, 115: 1760–1764, 1940.
9. Storck, A. H. Penetrating wounds of the abdomen. *Ann. Surg.*, 111: 775–819, 1940.
10. Taylor, F. W. Gunshot wounds of the abdomen. *J. Indiana M. A.*, 31: 342–345, 1938.
11. Warman, W. M. Shotgun wounds of the abdomen. *West Virginia M. J.*, 40: 169–172, 1944.
12. Zinninger, M. M. Penetrating wounds of the abdomen. *J. A. M. A.*, 124: 491–494, 1944.
13. Willis, B. C. Shotgun wounds of the abdomen. *Am. J. Surg.*, 28: 407–427, 1935.


DISCUSSION

Kellogg Speed (Highland Park, Ill.): This is most intriguing and interesting and reminds me of the experiments done by Sir Victor Horstley in the early part of the first World War. If you remember, he was a very eminent English surgeon who was sent to Mesopotamia. There he developed some experiments on gunshot wounds of the skull and brain. He took some crania and filled them with various solutions and mixtures and applied gunshot injury to them, working out some mechanical principles and two tables, the inner table much more widely scattered. He took photographs of the track through the brain tissue and other substances that he put inside the skull and was able to demonstrate some of this wide area of contusion and vibration. For that reason, the surgery of gunshot wounds of the skull was modified considerably in the early stages of that work. Those experiments are classical and are well worth going over again.

# Symposium on Skeletal Trauma

## THE LOCAL PHYSIOLOGY OF BONE REPAIR*

### WITH PARTICULAR REFERENCE TO THE PROCESS OF NEW BONE FORMATION BY INDUCTION

MARSHALL R. URIST, M.D. AND FRANKLIN C. MCLEAN, M.D.

*Los Angeles, California* *Chicago, Illinois*

STUDENTS of the problem of osteogenesis have known for centuries that fracture healing begins by proliferation of cells of periosteum and endosteum. The periosteal reaction is typical intramembranous bone formation, and the endosteal reaction appears to be appositional bone formation. In both instances the new bone clearly originates from cells which are linear descendants of osteoblasts.

Recent experimental investigations present strong evidence of the existence of another mechanism of osteogenesis described as the process of bone formation by induction. The theory of induction is a likely solution of the mystery of how the new bone is guided toward the fracture lines, not haphazardly in all directions, but directly out in the fracture gap, sometimes for long distances along devious routes in order that the bony deposits from one fragment will fuse with the bony deposits on the other fragment to produce union of the fracture. The object of this report is to define the concept of induction, and to summarize the experimental evidence that an induction system is developed in connection with the healing of fractures.

The anatomy of bone repair is well known and need not be reviewed here except to emphasize a few important points which have been established and confirmed by many laboratory investigations and clinical reports.[6–12] The development of various kinds of connective tissue cells and the differentiation of cartilage and bone occurs in fracture healing in a standard pattern which is retained throughout the phylogenetic history, embryogenesis and postfetal growth of the skeleton. The organization of the process of bone repair is exactly the same as in fetal osteogenesis. In each case a model of the bone is first formed from young fibrous connective tissue and cartilage. The model is then invaded by blood vessels, perivascular connective tissue cells, and osteoblasts from periosteum and endosteum and replaced by new bone.

The new bone formation begins at some distance from the fracture line, originating in the subperiosteal and subendosteal spaces, and grows only by extension from these regions, and never at random in the fracture gap.

The formation of a fibrous, fibrocartilaginous, or cartilage model is always a preliminary step and represents an indispensable part of the process of bone repair. The size and shape of the model conforms to the proportions of the fracture gap and the displacement of bone ends. Even in impacted fractures through cancellous bone with hardly any bone defect, a microscopic plate of fibrous tissue or fibrocartilage is formed between the bone trabeculae at the line of the injury. In displaced fractures of the shaft of the long bones, the defect becomes filled with a mass of fibrocartilaginous callus which early re-establishes the continuity of the original bone. An irregular type of endochondral ossification and replacement of the cartilage by bone follows by means of a special design of construction. In cross section this

* From Wadsworth General Medical and Surgical Hospital, U. S. Veterans Administration, Sawtelle, Los Angeles, Calif., and The University of Chicago, Department of Physiology, Chicago, Ill. Acknowledgment of grants-in-aid received by the authors has been made in each of the papers reviewed. Further acknowledgment is due to the Division of Research Grants, National Institutes of Health, Public Health Service and to the Josiah Macy, Jr. Foundation, who supported the investigations dealing with induction.

resembles the process of "cantilevering out" used for the construction of a fixed-arch bridge. The new bone grows out upon the surface of the model enveloping the cartilage to form an arch of new bone over the fracture gap. Like ribs and spandrels which are let down from the arch of a bridge to suspend the deck, the new bone grows through and replaces the cartilage centripetally toward the fracture gap. The space between the fracture ends thus becomes filled with new bone which grows inside the arch in the final stage of healing.[9]

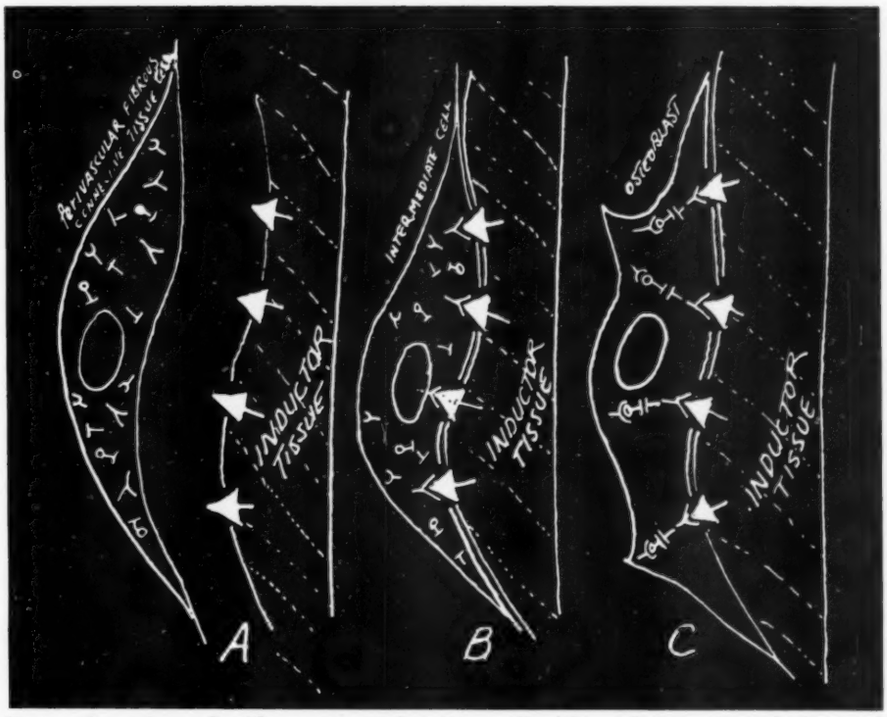



FIG. 1. A, perivascular fibrous connective tissue cell with symbols to show the internal molecular architecture of the cytoplasm and an inductor-tissue, e.g., cartilage, bone, urinary bladder epithelium. The surface of the inductor-tissue has a specific molecular architecture shown by the arrowheads. B, an induction system is developed when the surfaces of the young undifferentiated cell and the inductor-tissue come into contact. The surface-oriented molecules of the inductor (arrowheads) fit into specific (notch-shaped) molecules in the surface of the young cell like a key fits into a lock. C, the formation of an osteoblast from an undifferentiated connective tissue cell by induction occurs after this molecular segregation process continues and results in "trapping and fixing" of the internal molecules in a specific pattern. The changes in the architectural pattern of the surface and deep molecules produce the differentiation of an osteoblast from a young connective tissue cell by the process of induction. In this way induction is visualized as a physical-chemical process which requires contact of cells and tissues and is transmitted from one layer to another.

## OSTEOGENETIC POTENCY AND NEW BONE FORMATION BY INDUCTION

The definition of the term *induction* and the distinction between new bone formation by induction and new bone formation from tissues which have *osteogenetic potency* have been presented by Urist and McLean as follows.[12] The term osteogenetic potency describes the capacity of certain tissues to produce new bone. This potency is demonstrated by osteogenetic activity following injury or autogenous transplantation. The term *induction* designates the physical-chemical effect of one tissue upon another in contact with it. New bone formation by induction occurs from ingrowing perivascular connective tissue cells and is generally associated with absorption of bone or cartilage following injury or transplantation. The connective tissue cells which are *induced* to form bone by *induction* develop a potency which might otherwise have remained latent in the lifetime of the individual. (Fig. 1.)

New bone formation from each or both of

these mechanisms was observed by means of experiments in which samples of various parts of a healing fracture were dissected free and transferred to the anterior chamber of the eye.[12] The tissues found at the site of a fracture which conceivably might contribute to the process of bone repair are: (1) organizing hematoma, (2) fascia, (3) muscle, (4) periosteum, (5) cortical bone, (6) cancellous bone, (7) endosteum, (8) bone marrow, and (9) fibrocartilaginous callus. Each of these tissues was tested for the presence of cells with osteogenetic potency or for the *inductor* by means of the traditional method of transplantation of an autogenous sample to a soft tissue organ in which there is no possible contact with pre-existing bone.

*Organizing Hematoma, Fascia and Muscle.* These tissues did not produce new bone following transplantation and it is reasonable to conclude that they do not contain any cells which have osteogenetic potency. These transplants did not induce osteogenesis within a period of thirty days in the eye and thereby indicate that such tissues make no particular contribution to the process of bone repair.

*Periosteum.* Periosteum from young, growing individuals contained cells which evidently were linear descendants of osteoblasts and quickly produced new bone. Transplants of growing periosteum were *histogenetic*, produced simple intramembranous new bone formation, and later transformed into a mass of compact cortical bone. Transplants of periosteum from adult animals after growth of the skeleton was nearly finished did not produce new bone. Similar transplants of periosteum from adult animals within twenty-four hours after a fracture showed that the bone-forming potentialities of this tissue could be reactivated by an injury of the underlying cortex. The ability of the periosteum to become quiescent and morphologically indifferent, with return to the original bone-forming function after an injury, is an example of the phenomenon of *modulation*. The term "modulation," as distinguished from differentiation, describes reversible or temporary alterations in the function of cells.

*Compact Bone.* Cortical bone obtained from the fracture ends was carefully scraped free of periosteum and washed as free of bone marrow as possible. Transplants of this tissue examined by histologic methods, appeared completely devoid of *any living cells*, yet did produce a scant deposit of new bone in the eye in a few instances after a latent period of four weeks. The new bone appeared to arise not from any transplanted osteoblasts but from ingrowing perivascular connective tissue cells of the host which established contact with the donor tissue in the process of absorbing it.

*Cancellous Bone and Endosteum.* These tissues are inseparable from each other and from red bone marrow. A cancellous bone graft, therefore, constitutes a transplant of at least three different kinds of tissue. The net result of all three when transferred to the anterior chamber of the eye, was appositional new bone formation on the surface of the transplant, and progressive replacement of old bone tissue with new bone. It was not possible to determine whether the new bone formation was the result of proliferation of transplanted endosteal cells or whether cells of the host, by means of an induction system, were responsible for the new bone. It seems likely that both mechanisms were involved and that osteogenesis and "creeping substitution" were *sustained* by a specific affinity of the ingrowing cells of the host for the organic substance of the donor tissue, and that this affinity was produced by an *inductor* present in bone tissue.

*Bone Marrow.* Pure samples of bone marrow teased out of the medulla, from either fractured or unfractured bones, produced new bone formation. Here again it was not possible to determine whether the new bone developed from reticular cells of the marrow stroma which survived transplantation or from ingrowing cells of the host by induction. The end product of such transplants, however, was entirely different from that observed from osteoblasts in periosteum. Bone marrow produced not only bone tissue, but was also *organogenetic* and produced an entire ossicle which had a cortex, a marrow cavity, and hematopoietic bone marrow, and conformed in shape to the anterior chamber of the eye.

*Fibrocartilaginous Callus.* The most convincing evidence of the process of new bone formation by induction came from transplants of pure samples of fibrocartilage and hyaline cartilage. These tissues promptly produced a complete ossicle, which appeared histologically to develop mainly by induction. A scant amorphous deposit of new bone was produced (after a latent period) from tissues which were devitalized by boiling or freezing prior to transfer to the eye. However, samples which

were allowed to dry in a calcium chloride desiccator at room temperature invariably produced negative results. New bone formation under these conditions could develop only by some process such as induction. Negative results with dried tissue after autolysis of the cells at room temperature can be interpreted to support the idea that an inductor may be an enzyme.[5]

*Induction and Regeneration of Bone.* According to Spemann[4] induction plays a role in the normal course of events of development of all animal tissues and organs. The aforedescribed experiments present the evidence that the process of induction produces the replacement of fibrocartilaginous callus by bone and that an induction system directs the process of osteogenesis across the fracture gap. The physical-chemical mechanisms which may be involved have been investigated with many different kinds of tissues in the field of experimental embryology. In 1938 Spemann[4] presented a monograph which summarized his work which is now famous for having demonstrated the effect of the chordamesoderm upon the overlying ectoderm calling forth the formation of a neural plate, and the induction of lens in the epidermis overlying the eye vesicle. Quite a number of such induction systems were later discovered in other tissues.

Weiss[13] transplanted frozen-dried cartilage into the limbs of salamander larvae and noted deposits of new cartilage on the surface of the devitalized donor cells when the graft was absorbed. The new cartilage formed only in direct contiguity with the graft as though the cells of the donor communicated their state to their neighbors, which then passed it on further and so on down the line from a focal area like an infectious wave.[14] The morphologic picture of replacement of the fibrocartilaginous callus by new bone and the course of events which lead to bone repair have most of the features of induction as described previously.

All of the known examples of induction systems show that the process begins only if the inducing and the induced layers are in close contact. The mechanism was visualized: by Weiss as orientation of molecules on the surfaces of cells, with a high degree of specificity and complexity and with interaction of both physical and chemical mechanisms; by Spiegelmann[5] as the reactions of unique enzyme patterns.

## CALCIFICATION

The fracture callus is calcified in the same way and by the same mechanism as growing bone everywhere in the body. Calcification is an entirely separate, although normally concurrent, phenomenon in bone formation and appears to have no causal effect upon induction of osteogenesis. Calcification of bone begins in a specially prepared intercellular matrix, provided that calcium and phosphate ions diffuse in sufficient concentration from the capillary blood plasma. The cartilage matrix and fibrocartilaginous callus are calcified only at the line of contact with the osteogenetic front and proceeds just as it does at the epiphyseal plates through a zone of provisional calcification. Undecalcified sections of biopsy specimens from clinical fractures show a very slight lag in the rate of calcification of the callus under normal circumstances but this has no apparent effect upon the rate of healing and time of union.[9]

The changes which take place in the matrix of cartilage and bone prior to calcification, which differentiate a calcifiable from a noncalcifiable substrate are not known. The physiology of the calcification mechanism, the chemical composition and the crystalline structure of the bone salt are major problems in themselves for further investigation and have been reviewed elsewhere.[1]

*Alkaline Phosphatase, Calcium Ion and pH.* The callus, like all other tissues, in which ossification occurs, contains alkaline phosphatase. This enzyme has been demonstrated in the cytoplasm of osteoblasts, and is currently believed to be connected with the formation of collagenous fibers and the bone matrix, rather than the calcification mechanism. The role of calcium ion in the tissue fluids of bone, and pH of the fracture hematoma, and the alkaline phosphatase content of the procallus have been discussed or investigated in detail but hardly any effects were noted which could be considered characteristic of the process of bone regeneration. None of these factors has been correlated with the process of induction.

## SYSTEMIC FACTORS

The local process and the systemic factors in bone repair are, of course, inseparable and should so be considered regardless of the emphasis placed on the local factors in the

clinical management of fractures. The fracture callus obtains tissue-building materials from the systemic circulation just as does any other tissue. The importance of the systemic factors was not appreciated until it was found that the callus differs from other organs in that it received the highest priority on tissue-building materials. Furthermore, nearly all of the elements used in building bone matrix are present in muscle tissue, readily mobilized, and stored in a supply which is hardly exhaustible. These extraordinary interactions of the body reparative processes were observed in rats with multiple fractures which can heal twenty-two concurrent fractures or forty-four fractures incurred in series in the same period of time that they can heal a single fracture.[11]

Recent observations by Sissons and Hadfield[3] upon cortisone-treated rabbits indicate that systemic influences can produce an appreciable effect upon the local course of bone repair. The rabbit was found to be a species which is sensitive to cortisone, more like the human than the rat in which previous experimental studies showed hardly any significant effects upon bone repair. In rabbits treated with 10 to 20 mg. of cortisone per kilo, periosteal new bone formation was scanty, cartilage cells failed to hypertrophy, chondro-osteoid tissue encapsulated the fibrocartilaginous callus, and there was delay in union. These changes were similar to those previously described in rachitic rats,[7,8] and lower vertebrates[2] in which fracture healing is normally a slow, incomplete process and osteogenesis as it occurs in higher vertebrates seems not yet evolved. The formation of chondro-osteoid tissue indicates interference with the process of absorption of cartilage and apparently also with the mechanisms of new bone formation by induction.

## SUMMARY

The repair of bone always occurs by preliminary formation of a fibrous or fibrocartilaginous model of the fracture site. The cartilage model is replaced by new bone by (1) proliferating cells of periosteum and endosteum, (2) perivascular connective tissue cells which grow in from these areas by induction, and (3) a combination of these two processes. The process as a whole is an example of diverse means to a common end which is the union of the fracture.

## REFERENCES

1. McLean, F. C. and Urist, M. R. Newer knowledge of postfetal osteogenesis. *Bull. Hosp. Joint Dis.*, 12: 431–442, 1951.
2. Pritchard, J. J. and Ruzicka, A. J. Comparison of fracture repair in the frog, lizard, and rat. *J. Anat.*, 84: 236–261, 1950.
3. Sissons, H. A. and Hadfield, G. J. The influence of cortisone on the repair of experimental fractures in the rabbit. *Brit. J. Surg.*, 39: 172–178, 1951.
4. Spemann, H. Embryonic development and induction. New Haven, 1938. Yale University Press.
5. Spiegelmann, S. Differentiation as the controlled production of unique enzymatic patterns. *Symp. Soc. Exper. Biol.*, 2: 286–325, 1948.
6. Urist, M. R. and McLean, F. C. Calcification and ossification. I. Calcification in the callus in healing fractures in normal rats. *J. Bone & Joint Surg.*, 23: 1–16, 1941.
7. Urist, M. R. and McLean, F. C. Calcification and ossification. II. Control of calcification in the fracture callus in rachitic rats. *J. Bone & Joint Surg.*, 23: 283–310, 1941.
8. Urist, M. R. Calcification and ossification. III. The role of local transfer of bone salt in the calcification of the fracture callus. *J. Bone & Joint Surg.*, 24: 47–67, 1942.
9. Urist, M. R. and Johnson, R. W., Jr., Calcification and ossification. IV. The healing of fractures in man under clinical conditions. *J. Bone & Joint Surg.*, 25: 375–426, 1943.
10. Urist, M. R. Calcification and ossification in healing fractures. Conference on metabolic aspects of convalescence. Tr. Fourteenth Meet., New York, 1946. Josiah Macy, Jr. Foundation.
11. Urist, M. R. and McLean, F. C. Bone repair in rats with multiple fractures. *Am. J. Surg.*, 80: 685–695, 1950.
12. Urist, M. R. and McLean, F. C. Osteogenetic potency and new-bone formation by induction in transplants to the anterior chamber of the eye. *J. Bone & Joint Surg.*, 34A: 443–470, 1952.
13. Weiss, P. Perspectives in the field of morphogenesis. *Quart. Rev. Biol.*, 25: 177–198, 1950.
14. Weiss, P. Chemistry and Physiology of Growth, chap. VII, p. 157. Princeton, 1949. Princeton Univ. Press.

## QUESTIONS

Marshall R. Urist (Los Angeles, Calif.): This is the first question: In considering the induction concept what tissue is induced to form bone by the cartilage which was transferred to the anterior chamber of the eye?

The tissue which is induced was created by the host. The transplant attaches itself through an inflammatory reaction on the outer surface of the iris. The inflammatory reaction produces granulation tissue which encapsulates the donor tissue. Young perivascular connective tissue cells and new capillaries then grow into the implant and are induced to form new bone by *contact* with the inductor tissue. The inductor elicits a capacity in

these cells which otherwise may have remained latent for the lifetime of the individual.

The anterior chamber of the eye, a visceral organ, or any isolated soft part will provide ideal conditions for investigations of the process of new bone formation by induction. In a host bed in skeletal tissue, such as a bone defect, special cell potencies and cellular reactions enter into the process. When an inductor tissue is implanted into a fresh bone defect, the operation itself produces a proliferative reaction to injury by endosteal cells which have already inherited osteogenetic potency. The osteogenetic connective tissue cells, cells with inherited osteogenetic potency, appear to have a specific affinity for the substance of any tissue which contains the inductor. Thus in a bone defect the osteogenetic reaction appears not to occur from one of the other of two mechanisms, cellular potency, or induction, but by a combination of these two processes. Homogenous, heterogenous, or preserved bone transfers will not produce bone very readily and only after prolonged periods in the eye. However, these relatively weak sources of inductor will consistently induce new bone formation in bone-to-bone transfers where the host bed is able to initiate osteogenesis and sustain the process.

The second question has been submitted by Dr. Kellogg Speed: Would other contact material produce the same effect in the eye?

Our information to answer this question is very limited. Calcium phosphate salts, bone salts and apatite minerals have been tested with generally negative results. Other materials require further investigation.

There are four questions which deal with delayed union for which I would like to give one answer in order to stay within the space that has been reserved for osteogenesis. Does the theory of induction throw any light on the problems of delayed and non-union?

Yes, the concept of induction offers interesting possibilities for further work in this field. We are studying a series of ununited fractures in collaboration with Robert Mazet, Jr. and Paul E. McMaster. There is histologic evidence that in delayed union the induction system in the fracture site is incomplete and develops in irregular patches. In irreversible non-union there was complete arrest of the process of new bone formation by induction. The individual cases present a great many questions dealing with complications and treatment for which there is no one answer.

DR. FRANCIS MOORE'S presentation, "Normal Metabolic Sequences Following Trauma," described an interpretation of convalescent metabolism which will be published elsewhere at a later date.

# VASCULAR COMPLICATIONS AND SEQUELAE OF SKELETAL TRAUMA

LOUIS G. HERRMANN, M.D.
*Cincinnati, Ohio*

DR. MOORE has ably presented the metabolic processes which are important in the recovery of patients after trauma, and I have been asked to give you some idea of the clinical complications, particularly vascular complications, which follow serious trauma.

The seriousness of any wound of violence will depend to a great degree upon the kind as well as upon the extent of the damage to the underlying great blood vessels and mixed nerves. All of us have had impressed upon our minds many times the absolute necessity for immediate immobilization of injured limbs with fractured bones in order to reduce the damage to adjacent large blood vessels and thus prevent the more serious complications of hemorrhage, shock or loss of function of the extremity.

Severe loss of blood or extensive trauma to tissues of an extremity may produce widespread arteriolar spasm to compensate for the reduction in blood volume from extravasation of plasma or whole blood into the soft tissues of the affected extremity. When this vasomotor reaction is intense, a state of traumatic shock exists, and restoration of the blood volume by the intravenous administration of 5 per cent dextrose in water, blood plasma or whole blood becomes essential. It is true that peripheral vasoconstriction is primarily a protective reaction to severe injury of a limb; but when the vasospasm is intense, extensive and prolonged, it is certain to cause irreversible changes in muscles, nerves and, lastly, in the skin of the affected extremity.

Injury to the outer wall of a large artery or extensive trauma adjacent to a large artery often results in segmental arterial spasm, the so-called stupor arterielle, with partial or complete occlusion of the affected large artery. Reflex spasm of an entire major artery is difficult to demonstrate, yet we frequently observe profound spasm of the arterioles after trauma to an extremity.

Secondary arteriolar spasm may lead to thrombosis of the small arteries, with resulting ischemic necrosis of the muscles supplied by those arteries. The subsequent fibrosis and contractures give rise to the clinical entity which was originally described by Volkmann. More recently, Leriche, Griffiths and Scott have described this syndrome in detail.

Marked arterial insufficiency may also be brought about by compression or occlusion of large arteries from the accumulation of blood beneath rigid fascial coverings like those of the popliteal space. Tightly applied bandages or casts and the prolonged or improper application of a tourniquet may give rise to serious degrees of ischemia. Tourniquets should not be applied to injured extremities, but external hemorrhage should be controlled by digital pressure at the site of the bleeding, local pressure with gauze over the bleeding wound, digital compression of the principal artery to the part, or the elevation of the bleeding part to a point well above the level of the heart.

Absence of peripheral pulses in an extremity does not necessarily indicate that the main artery has been severed. Arteriosclerosis obliterans or congenital absence of large pedal arteries may be the cause. After severe trauma, however, absence of peripheral pulses is more likely to be the result of segmental arterial spasm or arterial thrombosis. If the extremity shows signs of arterial insufficiency, every means of stimulating the flow of blood through the collateral arterial pathways should be instituted without delay. We do not believe that vasodilating drugs or autonomic nerve blocking agents should be used in such patients, since they produce widespread vasodilatation in the normal parts of the body and cause little or no improvement in the arterial circulation of the ischemic extremity. Novocainization of the regional sympathetic nerves to the affected extremity may be of distinct value, but in our experience the mechanical stimulation of the collateral arterial circulation by passive vascu-

lar exercises still stands as the most effective method we have ever used.

The immediate management of an ischemic extremity is of utmost importance. Further mechanical, thermal or chemical trauma to the affected part must be avoided. The limb should be wrapped in cotton batting and kept on a soft pillow. Added heat must *never* be applied to an ischemic extremity, since heat only increases the local metabolism of the tissues and deprives them of their oxygen more quickly without greatly increasing the flow of arterial blood to the damaged tissues. Gangrene can be precipitated by such application of heat. Local cooling or refrigeration should be applied only to those parts which are irreparably damaged and which will be amputated when the patient's general condition has improved sufficiently to warrant operation.

The resting position which affords the best exchange of blood in an injured limb is slightly *below* the level of the heart. Edema which develops after injury may require short periods of elevation of the injured limb, but *prolonged* elevation of the part is harmful, particularly in patients past middle life. Ischemia of the tissues with gangrene of the skin of the toes has occurred many times in elderly patients whose otherwise uncomplicated fracture was treated by fixation in an unpadded cast and the extremity elevated high above the level of the heart.

Venous thrombosis is another common sequela of skeletal trauma, and early recognition of this complication is essential to successful treatment. The extension of the intravenous clot or the transportation of parts of a blood clot to the lungs or some other vital part of the body, constitutes the greatest hazard to life. Extensive or progressive thrombosis of the veins of an injured extremity may cause ischemic necrosis with autolysis of muscles and subsequent complete loss of function due to fibrosis. Propagation of blood clots in a proximal direction may lead to fatal pulmonary embolism. It is now the general consensus that the prophylactic ligation of the superficial femoral vein does not prevent the occurrence of fatal pulmonary embolism. The ligation of large veins to halt temporarily the proximal extension of a blood clot in the deep veins of the legs may be necessary occasionally to save the patient's life. The proper administration of adequate amounts of anticoagulants, heparin and dicumarol, has proved to be the most effective means yet devised for controlling this process of abnormal intravascular clotting of blood. The administration of these drugs must be controlled by daily studies of the clotting ability of the blood by the accepted laboratory methods. Serious and sometimes fatal hemorrhage may result from the misuse of these potent anticoagulant substances.

Areas of spreading *superficial* venous thrombosis which follow in the wake of trauma do not respond well to anticoagulants. Pulmonary embolism is rare after this type of venous thrombosis, but the local pain may be severe and the period of disability rather long. We have recommended the complete excision of such areas of superficial venous thrombosis or phlebitis by the method of phlebectomy under local anesthesia. This procedure reduces the period of disability to a few days, while weeks or even months are usually necessary for the spontaneous absorption of such blood clots. We have proved that these areas of venous thrombosis are not the result of bacterial action. Consequently, antibiotic substances are not indicated in the management of this common complication of skeletal injuries.

*Fat Embolism.* Trauma to long bones with or without fractures may cause the liberation of large quantities of liquid fat into the venous circulation. Recently Musselman and his associates have commented upon the frequency of fat embolism in injured persons. They have emphasized that the fat embolism may be fatal, and its presence should be suspected in injured patients who show a dusky cyanosis of the skin, mild shock unaccounted for by the injury, fever and the appearance of fat droplets in the urine or sputum. We have found that the intravenous administration of an alcohol-dextrose mixture (5 per cent ethyl alcohol in 5 per cent dextrose solution) is an effective means of clearing the pulmonary capillaries of fat droplets. This mixture also facilitates the emulsification and metabolism of the fat. The therapeutic dose of ethyl alcohol for man ranges between 0.5 and 1.5 cc. per kg. of body weight. The toxic dose of ethyl alcohol is between 8.0 and 15.0 cc. per kg. of body weight.

*Neurocirculatory Phenomena.* Injury to the bones of an extremity and particularly to the small bones of the hands and feet, either with or without fracture of these bones, may give rise to one of several complicated neurovascular syndromes. Much of the confusion which now

exists regarding these important consequences of trauma is directly due to the paucity of our knowledge concerning the associated pathologic physiology as well as the mechanisms which produce them. Homans has attempted to classify all of these disturbances under the single term of "minor causalgias," while DeTakats merely calls them "reflex dystrophy of the extremities." We still prefer to classify them under the following three headings:

*Causalgia.* True causalgia as described by Wier-Mitchell, usually results from injury to large mixed nerves with or without fracture of the long bones. The burning pain in the distal parts of the extremity is usually accompanied by vasomotor changes in the form of varying periods of vasoconstriction followed by vasodilatation. Sympathetic denervation of the part (thoracic ramisection or lumbar ganglionectomy) may be effective in reducing the intensity of the symptoms, but the relief of pain is not always complete. No specific treatment has yet been devised, consequently symptomatic and supportive measures must be relied upon when sympathectomy fails to bring about relief of pain. Psychotherapy has been advocated by many students of this syndrome.

*Phantom Limb Pain.* The severance of an extremity by shearing or crushing often leads to the development of constant, severe, and sometimes agonizing pain which is projected into the part of the body which had been removed. For many years it has been our opinion that this syndrome is the result of abnormal stimulation of afferent pathways in large mixed nerves of an extremity, by inflammation, compression, laceration or fibrosis of centrally conducting axones. We believe that much of this kind of pain can be prevented by special care of mixed nerves at the time of the original amputation. Large mixed nerves should be handled carefully and never clamped with forceps. A non-absorbable ligature of medium size should be applied about the uninjured nerve and then tied tightly about 1 inch proximal to the level of the amputation. The nerve distal to the ligature should be cut across squarely with a sharp knife about 5 mm. from the ligature. Large mixed nerves should not be injected with novocaine, alcohol or similar substances. The simple method of ligating the mixed nerve has greatly reduced the incidence of phantom limb pain in our experience.

*Post-traumatic Painful Osteoporosis.* We believe this syndrome should be considered as a distinct clinical entity. It is characterized by vasomotor instability with color changes in the fingers or toes, edema of the soft tissues and constant, severe visceral pain. This syndrome usually develops several weeks after trauma to the extremity.

The characteristic roentgenologic changes in the bones of the extremity may be described as spotty demineralization. This is entirely different from the atrophy of disuse which we commonly observe after skeletal injuries. From long experience in the management of this condition, we have become convinced that the widely misused and frequently discredited operation of periarterial neurectomy is still the best method of arresting the destructive changes in the bones, for reducing the swelling of the parts and for shortening the period of disability. It must be carefully and completely done in the early stages of the syndrome.

*Arterial and Arteriovenous Aneurysms.* Finally, we must keep in mind that direct injury to large arteries may result from compression against the shaft of the bone, punctured by spicules of the fractured bones, stretching from sudden dislocation of joints or soft tissues. The faulty placement of metallic pins or screws to hold fragments of long bones in place also takes a toll of limbs because of secondary and many times unsuspected injury to some large artery. Aneurysms of various types are occasional complications of skeletal injuries.

When large arteries are encased in dense fibrous sheaths like in the popliteal space, the sudden dislocation of the knee joint may shear the popliteal artery. Any accumulation of blood in the popliteal space warrants immediate surgical exploration of the area and the decompression of the space. If the artery is damaged, attempt should be made to repair it; but if the vein is injured, it should always be ligated. Serious and sometimes fatal pulmonary embolism may follow an attempt to repair a large vein.

Pulsating hematomas occur when the major artery has been lacerated. When a large artery has been completely severed, considerable internal hemorrhage usually results; but contraction of the cut ends of the artery favors thrombosis and complete occlusion, consequently aneurysms do not usually develop. Tangential injuries to large arteries always give rise to pulsating hematomas which later

develop into simple arterial aneurysms. If a spicule of bone or any foreign object should penetrate both the artery and vein, a pulsating hematoma may develop at first; but this rapidly assumes the clinical characteristics of an arteriovenous aneurysm.

The treatment of simple aneurysms depends upon the location and the kind of arterial aneurysm which has developed as a result of the vascular injury. The saccular aneurysms can usually be excised and the major artery reconstructed by lateral or end-to-end suture technics. Fusiform aneurysms usually require excision and replacement of the damaged segment of the artery by an autogenous venous graft.

Simple aneurysms and pulsating hematomas which do not cause ischemia of the extremity should be treated conservatively for many weeks, but those which cause marked ischemia in the distal parts of the affected extremity must be treated immediately by surgical exploration of the area and repair of the injured artery whenever that is possible. Delay will only give rise to irreparable changes in the soft tissues of the extremity due to the ischemia.

Arteriovenous aneurysms should be treated conservatively for several months before definitive surgical treatment is undertaken unless the strain on the heart is so great that impending cardiac decompensation exists. Ligation of the proximal vein may relieve the load on the right heart temporarily and give time for the development of a collateral arterial circulation.

REFERENCES

1. Griffiths, D. L. Volkmann's ischemic contracture. *Lancet*, 2: 1339–1344, 1938.
2. Herrmann, L. G. Phlebectomy in the treatment of acute thrombosis of saphenous varices or veins. *Arch. Surg.*, 64: 681–685, 1952.
3. Herrmann, L. G. Effect of dextrose-alcohol mixture upon pulmonary fat embolism. *Proc. Soc. Exper. Biol. & Med.*, 30: 558–559, 1933.
4. Herrmann, L. G. and Gibbs, E. W. Phantom limb pain: its relation to the treatment of large nerves at time of amputation. *Am. J. Surg.*, 67: 168–180, 1945.
5. Homans, J. Minor causalgia: a hyperesthetic neurovascular syndrome. *New England J. Med.*, 222: 870–874, 1940.
6. Leriche, R. Physiologie Pathologique et Chirurgie des Artères. Paris, 1943. Masson & Cie.
7. Mitchell, S. W., Morehouse, G. C. and Keen, W. W. Gunshot Wounds and Other Injuries of Nerves. Philadelphia, 1864. J. B. Lippincott.
8. Musselman, M. M., Glas, W. W. and Grekin, T. D. Fat embolism: a clinical investigation. Presented before The Central Surgical Association meeting, Toronto, March, 1952.
9. DeTakats, G. Reflex dystrophy of the extremities. *Arch. Surg.*, 34: 939–956, 1937.

QUESTIONS

Louis G. Herrmann (Cincinnati, Ohio): The first question which has been presented to me is as follows: what do you think of maintaining a tourniquet over long periods, one to two hours, during surgery?

Whenever possible, I prefer to do surgical operations upon the extremities without using a tourniquet. Fortunately, however, human extremities can withstand long periods of ischemia without untoward results. In my discussion, I referred to the harmful effects of a tourniquet when there was associated extensive venous stasis in the injured extremity. If an extremity can first be drained of blood by elevation and then a pneumatic type tourniquet or Esmarch bandage applied above the injured area, I do not believe serious complications will result from the ischemia that lasts only one or two hours.

The second question is as follows: Do you use anticoagulants in the presence of fresh bone injuries?

I believe anticoagulants can be used safely in such a patient if at least six hours has elapsed between the injury and the administration of heparin. We prefer the use of heparin in such patients because its anticoagulant effect can be controlled easily or abolished if hemorrhage becomes a complicating factor. It has been my conviction for many years that extensive posttraumatic venous thrombosis is the result of an increased coagulability of the blood; therefore, relatively small doses of heparin, such as 50 mg. intravenously every four hours or 100 mg. intramuscularly every six to eight hours, should prevent this dreaded complication of injury to an extremity.

The third question deals with established venous thrombosis and reads: In a patient with definite thrombophlebitis of the lower extremity postoperatively, with pain, swelling and increased heat, do you advise venous ligation? If so, what veins and at what level?

If the venous thrombosis began in the veins of the calf muscles and if there is evidence of extension into the veins of the thigh, ligation of the superficial femoral vein in Scarpa's triangle of the thigh might limit the venous thrombosis to the leg and thigh. Since venous thrombosis of this type is undoubtedly due to abnormal changes in the blood and not due to local disease in the veins, I believe anticoagulants should be used simultaneously. I see no harm which would follow ligation of the superficial femoral vein; while, on the

other hand, there is no scientific evidence that the incidence of pulmonary embolism has been reduced by that procedure. We prefer to use anticoagulants alone in the management of such peripheral venous thrombosis. The symptoms will subside with bed rest, local warmth to the part and elevation of the extremity. If the acute venous thrombosis is limited to the saphenous veins, I believe the period of disability will be greatly reduced if the saphenous veins, including the clots, are excised.

Rapidly ascending venous thrombosis of the deep veins of the extremity is the only real indication for ligating the superficial femoral vein.

The fourth question is: Do you recommend excision of a spastic section of an artery in impending Volkmann's contracture?

My answer to this specific question would have to be "No." I believe Volkmann's contracture is actually the result of thrombosis of the arterioles in muscles and skin and not due primarily to the vasospasm. It is usually too late to relieve the vasospasm after the clinical picture of Volkmann's contracture has become established. The autolysis of muscles takes place very rapidly after arteriolar thrombosis and the color changes in the skin or the contractures are late effects of the vascular insufficiency. If a segment of artery appears spastic at the time of operation immediately after injury, denudation of the adventitia of the artery and infiltration of 2 per cent novocain solution about the artery should cause the segment to relax.

The final question deals with fat embolism and is as follows: Would you comment on the diagnosis of fat embolism and its treatment?

Fat embolism must exist much more frequently than any of us think. We have evidence that most individuals who are seriously injured, particularly those who have injury to long bones of the extremity, do have varying degrees of fat embolism. The question is: When does that fat embolism become of clinical significance or of serious nature? The presence of fat in the sputum and in the urine certainly indicates that a lot of fat is being trapped in the pulmonary circulation and renal circulation. It is our belief that severe degrees of fat embolism with dusky cyanosis of the skin of the extremities, with fever and shock which is not accounted for by the injury should be treated by active means of overcoming the fat embolism.

I spoke to you about the use of dextrose-alcohol solution intravenously. In our experience, it is still the most effective means of reducing the surface tension of the fat, thus permitting it to go through the pulmonary capillaries and through the small capillaries of the kidneys. Fat may then appear in the urine, in the sputum and in the gastrointestinal tract. I still think that the dextrose-alcohol mixtures offer a very effective means of combatting the more serious degrees of pulmonary as well as cerebral fat embolism. All patients with serious trauma to extremities must have varying degrees of fat embolism.

# COMPLICATIONS AND SEQUELAE OF INFECTION IN SKELETAL TRAUMA*

OSCAR P. HAMPTON, JR., M.D.
*St. Louis, Missouri*

INFECTION is a dreaded complication in all fields of surgery. When it has become established, death may follow or, if this be avoided, morbidity is increased and a high degree of permanent disability may follow. In the field of musculoskeletal trauma complicating infection is unlikely to be fatal but its ravages may necessitate amputation or produce a severely handicapped or worthless extremity.

Wound infection on a clinical basis may be classified as: (1) Invasive infection; this results from the bacterial invasion and destruction of living tissue such as occurs in true gas gangrene. (2) Wound suppuration; this results as a localized process from the septic breakdown of dead and devitalized tissue including blood clot in dead space. In effect, this is abscess formation. It is the most common type of wound infection in musculoskeletal surgery. (3) Surface infection; this results as a localized process from the septic breakdown of wound exudate from raw surfaces of open wounds, granulating surfaces and burns. This clinical classification of wound infection conforms in substance to that suggested previously by Churchill in a discussion of war wounds.

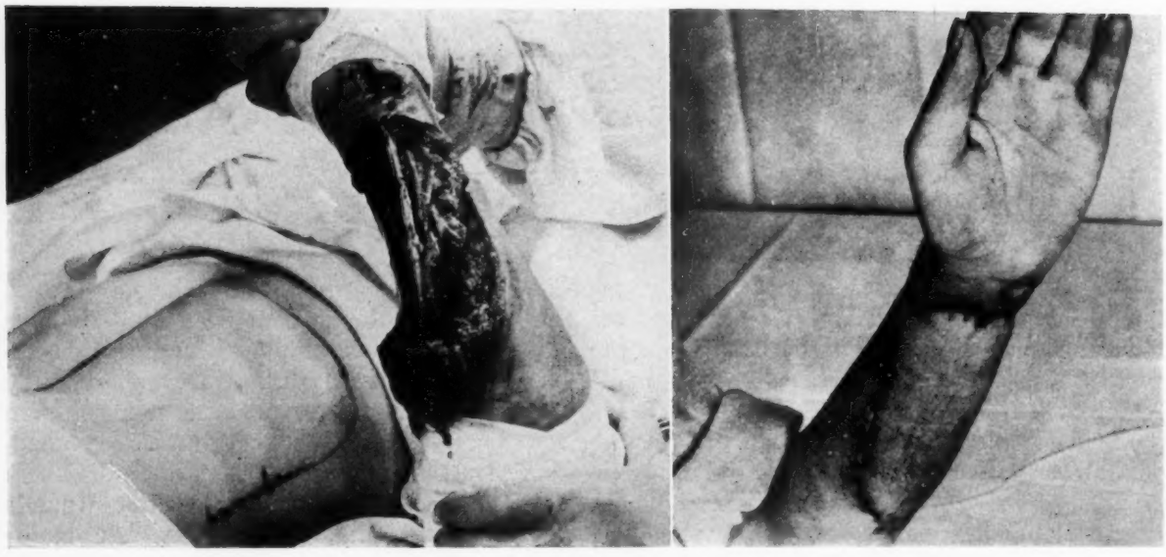
The complications and sequelae of wound infection may be considered as systemic and local. With invasive infection, both reactions are prominent. There is considerable local death of tissues in the wound and, of more importance, a systemic toxemia which can be fatal. In gas gangrene, an example of true invasive infection, previous living muscle tissue is killed by bacterial action and there is marked toxemia which produces altered sensorium, high fever, severe secondary anemia, etc. Unless therapy is prompt and effective, death follows. Other invasive infections may be equally serious. In spite of antibiotic coverage, symbiotic infection by hemolytic cocci and facultative anerobes can cause extensive death of tissue locally and severe systemic toxemia with falling red cell count, hemolytic icterus, edema and prostration. Death may occur. Even though the patient may survive such invasive infections, amputation may be necessary or only a badly crippled extremity may remain. Fortunately, they are not common complications of skeletal trauma in civilian surgery although open fractures are more likely to be complicated by gas gangrene than wounds of soft parts only, except when the major vascular supply has been interrupted.

Wound suppuration and surface infection, however, are relatively common complications. The former may occur whenever contaminated dead tissue including blood clot in dead space, the pabulum of sepsis, remains in a traumatic or surgically produced wound. Unfortunately, either unavoidably or as a result of inadequate or inexpert surgery these conditions are too often fulfilled. Surface infection is a not uncommon complication because in the field of musculoskeletal surgery open wounds advisedly or inadvisedly often persist. Contamination of the exudate on the raw surfaces of a persisting open wound is inevitable and with it, a superficial type of wound suppuration develops which delays or prevents healing.

The complications and sequelae of wound suppuration and surface infection are principally local rather than systemic. Of course, there may be some toxemia with febrile reaction and, with chronic sepsis, amyloid disease can occur. With large open wounds, particularly extensive granulating surfaces, considerable plasma loss occurs leading toward hypoproteinemia. Secondary anemia, loss of appetite, malnutrition, etc., usually follow. Septic phlebitis with its well known systemic effects can occur. This discussion, however, is concerned principally with the localized effects of a suppurative process in a wound involving musculoskeletal tissue.

Wound suppuration, either deep or surface, develops in spite of antibiotic protection. These valuable agents, while effective in preventing

* From the Department of Surgery, Washington University Medical School, St. Louis, Mo.



1A 1B

FIG. 1. A, large open wound of forearm with many tendons exposed and dying nine days after injury ready for coverage with pedicle graft. B, completely healed wound several weeks later. Prompt coverage of exposed collagenous tissues prevented their death and was an important step in salvaging some use of the hand.

or minimizing osteomyelitis, suppurative arthritis, brain and lung abscesses, etc., remote from the site of a wound infection, will not sterilize dead and devitalized tissue remaining in a wound including blood clot in undrained dead space and, therefore, will not prevent wound suppuration. Antibiotics moreover will not inactivate the proteolytic enzymes in undrained pus. The pus which forms gravitates into all of the crevices of the wound and bathes all the exposed tissues. Proteolytic enzymes in the pus gradually necrotize certain of the living tissues thereby creating additional devitalized tissue which, in turn, decomposes into more pus. Thus a vicious circle is established with persisting suppuration and continuing necrosis of living tissues. This is really the scourge of wound suppuration.

The degree and extent of death of living tissue produced by the proteolytic action of pus varies with the circumstances. When there is pocketing, puddling and pooling of pus in a wound, the destruction of tissue is likely to be extensive. It is maximum when wound suppuration occurs in a sutured wound and the pus remains trapped and spreads underneath the closed epithelial bridge. It is minimum and perhaps nil when the wound is well drained so that a minimum amount of pus if any remains in contact with the living tissues. Since the most effective drainage may be provided through a wide-open wound, many traumatic wounds are intentionally left unsutured in an effort to minimize the chances and effects of wound suppuration. The open wound, however, is not without undesirable consequences.

When a fresh wound is left open, tissues deep to the skin are exposed. Muscle tissue survives such exposure splendidly but tendon, fascia, ligament, cartilage and bone cortex, tissues which collectively may be called collagenous tissues, do not survive when left exposed. Necrosis gradually ensues partially at least from lack of nourishment. In addition, surface infection sets in and the necrotizing enzymes in the pus accelerate the death of these exposed tissues. The combination renders certain the loss of these collagenous tissues if they remain exposed in an open wound for more than a few days even though the original wound was well débrided and, therefore, in the beginning free of dead tissue and wound suppuration.

The same tissue susceptibility is found deep in a wound in the presence of suppuration. Again, muscle tissue appears to resist the necrotizing effect of enzymes in pus but the collagenous tissues cannot. Even though they are adequately covered by muscle or skin and, therefore, not exposed in the wound, tendon, facia, ligament and cartilage undergo necrosis when they remain for some time in contact with purulent material. Bone cortex also fails to survive although probably less from proteolytic enzyme action as will be discussed below.

The step-by-step effects of exposure or persistent bathing in purulent material on tendon, fascia, ligament and cartilage are comparable. Tendon soon loses its smooth glistening appearance. There is gradual softening and loss of definite margins. Finally, there is complete disintegration and loss of continuity. Stringy ends remain as avascular tissue undergoing suppuration and prolonging the suppurative process in the wound. The time required for a complete disintegration of tendon varies of course. It has been shown that a tendon can be salvaged after it has shown some softening but still remains as a definite structure if the suppurative pabulum in the wound can be eliminated and the tendon successfully covered with healthy soft parts. As a rule this must be achieved within less than a week or the tendon is doomed. (Figs. 1A and B.)

Fascia reacts in a comparable fashion. It disintegrates more rapidly, however, and leaves multiple strings of devitalized material. Fortunately, the loss of fascia is seldom highly significant and it may be extensively sacrificed during débridement in order to eliminate it as a focus of suppuration. Ligament shows more resistance but slowly succumbs. Its loss, of course, is usually of significance and it cannot be excised as freely at débridement.

Articular cartilage when left exposed or covered with pus soon loses its glistening appearance and softens in its more superficial layers. Finally, full-thickness death occurs and large plaques become separated from the subchondral bone. (Figs. 2A and B.)At this stage the joint is doomed and a fused joint in optimal position with a healed wound is the best possible result. (Fig. 2C.) So favorable a result, however, is by no means certain. The septic process may go on and on with extensive destruction of bone and surrounding soft tissues. Abscesses may extend up fascial planes. Amputation or even death are not remote possibilities particularly if a joint of the lower extremity is the one involved.

A peculiar reaction of articular cartilage to prolonged contact with pus is that, while it becomes separated from its subchondral bone, the large separated plaques then dissolve quite slowly and instead serve to prolong the suppurative process. It is because of these cartilage sequestra in a suppurative joint, at least in part, that wide exposure and removal of all dead and purulent material is essential if the suppuration is to be eliminated. While articular cartilage is susceptible to the proteolytic ferments in pus, full-thickness loss does not occur quickly and the joint usually may be salvaged if the suppurative process can be eliminated and the cartilage adequately protected by closing the joint within five to ten days after it began.

FIG. 2A. Areas of articular cartilage on distal end of femur which have undergone digestion by proteolytic enzymes in pus which collected in the knee joint from an adjacent septic fracture.

The problem in bone is on the same basis but it is more complex. Experimental observations in dogs have shown that the outer third of the cortex of the shafts of long bones is nourished by the periosteal blood supply. If periosteum is stripped from bone, the outer cortex will die. If there is no subsequent sepsis and the bone is promptly covered with reasonably well vascularized soft parts and wound healing obtained, they will re-adhere and the dying bone will be replaced by new bone as a part of a single process so that, clinically, death of bone is not recognized. If, however, the bone cortex remains uncovered or if sepsis develops so that re-attachment of soft parts does not occur, sequestration follows.

The skeletal traumas which may be followed by wound suppuration include open fractures and open surgical procedures on bone. In each instance some bone cortex is denuded of its coverings as periosteum is stripped from it. In both, early and successful coverage of denuded bone with healthy soft parts is highly desirable.

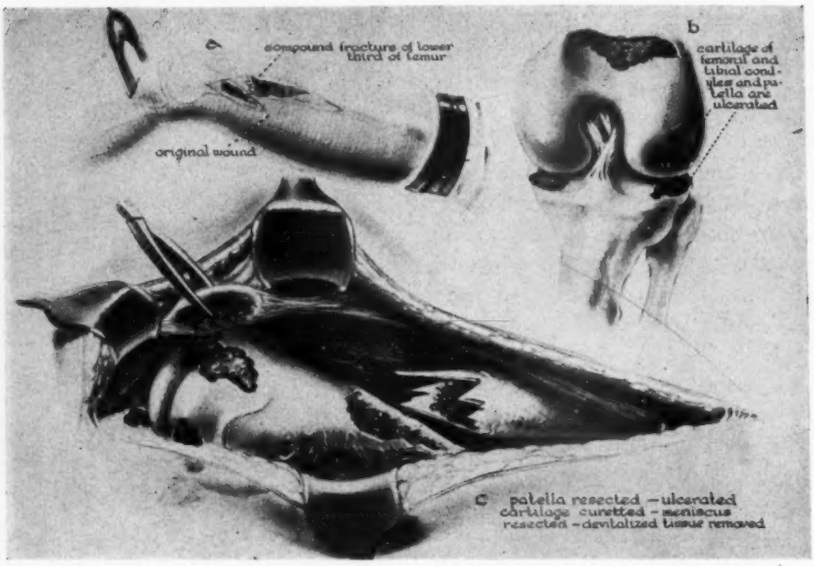

a
compound fracture of lower third of femur
original wound
b
cartilage of femoral and tibial condyles and patella are ulcerated
c patella resected—ulcerated cartilage curetted—meniscus resected—devitalized tissue removed

FIG. 2B. Artist's drawing of knee of another patient which shows considerable destruction of articular cartilage due to enzymes in undrained pus.

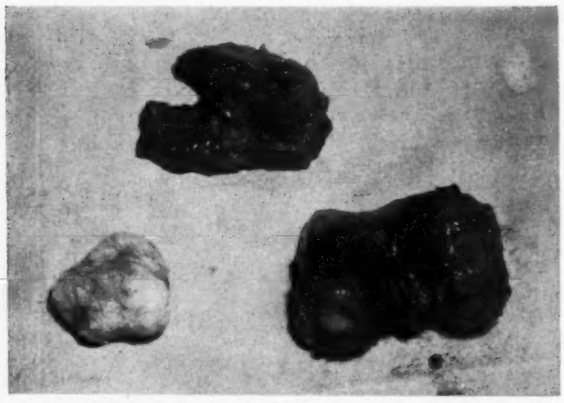
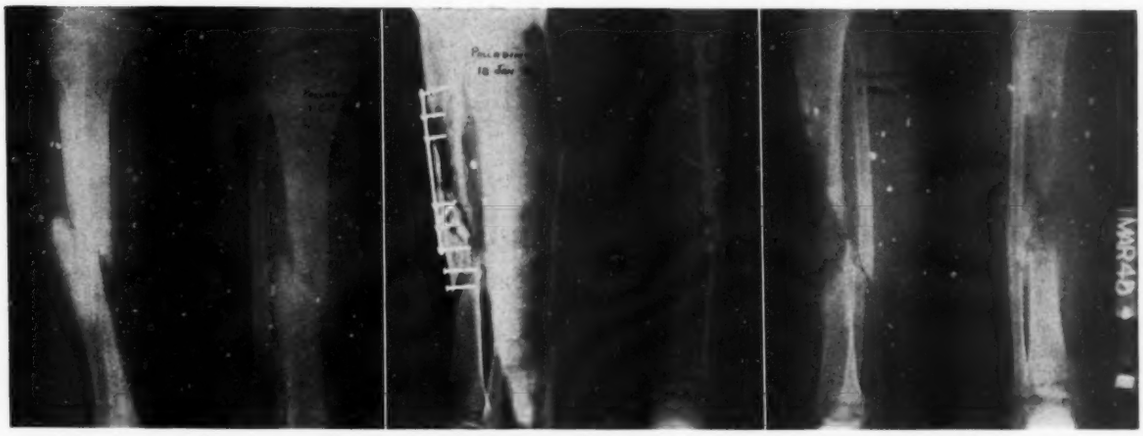


FIG. 2C. The necrotic articular surfaces of the femur, tibia and the patella excised at resection of a knee for a severe suppurative arthritis which began as an apparently innocuous open fracture of a tibial condyle.

This may be achieved, of course, by wound closure which, however, denies the advantages of open drainage and will enhance the chances of wound suppuration particularly if débridement is inadequate or if the closure is with excessive tension or over unobliterated dead space. If wound suppuration occurs, soft parts either muscle or skin do not re-adhere to bone and, as outlined previously, the denuded bone will not survive. The surgical problem, therefore, is to avoid wound suppuration and, yet, to obtain wound healing promptly so as to salvage the denuded bone.

With wound suppuration the death of bone unfortunately may not be limited merely to the devitalized external bone cortex. When persistently bathed in pus, the fine trabeculae of cancellous bone lining the marrow cavities usually are destroyed. As a result there may be considerable endosteal necrosis up and down the marrow cavity. Loss of both endosteal and periosteal blood supply together with thrombosis of vessels in the haversian systems which may follow due to the septic process will lead to death of considerable bone and massive sequestration. (Figs. 3A, B and C.) This is one of the more dreaded sequelae of infection following skeletal trauma. The loss of bone may prevent union, permit only a non-functioning precarious union or be so extensive as to establish a segmental defect. Even after wound healing these deficiencies often cannot be overcome readily by bone grafting because of the scarred, unhealthy soft tissue beds in which the bones lie. Amputation often must be the procedure of choice. It seems worth while to point out that the extent of the necrosis of bone which complicates wound suppuration is usually in direct ratio to the speed with which the condition is eliminated by re-débridement of remaining dead tissue in the wound and effective drainage.

Of course, sequestration of bone does not always occur in an infected fracture but wound suppuration, even without sequestration, at best usually delays or prevents the healing of fractures. The formation of soft callous may not occur or if it does, it is susceptible, as other collagenous tissues, to digestion by proteolytic enzymes. The bone ends, therefore, are not



3A 3B 3C

FIG. 3. A, roentgenograms of oblique fracture of both bones of the leg in the middle third. B, roentgenograms showing massive sequestration of bone which followed sepsis in the wound through which open reduction and internal fixation were performed. The operative procedure apparently entailed considerable periosteal stripping. C, massive segmental defect of bone following removal of metal and sequestra. Compare with Figure 3A.

sealed together by normal healing processes. Instead, unhealthy granulation tissue and later scar between the fragments slowly form and with them goes delayed or non-union of fractures and all the associated functional and economic loss.

## SUMMARY

1. Even under antibiotic protection invasive infection by clostridia and hemolytic cocci with facultative anerobes may occur. Death may follow or amputation may be necessary. Antibiotic protection, moreover, will not sterilize dead and devitalized tissue including blood clot in undrained dead space and, therefore, it will not prevent wound suppuration, deep or surface. It will not, moreover, inactivate the proteolytic enzymes in undrained pus.

2. The important effect of persisting wound suppuration is death of previously living collagenous tissues. Tendons become totally destroyed and lose continuity leading to permanent loss of function. Articular cartilage becomes necrotic and acts as a focus of persisting suppurative arthritis leading to total destruction of the joint and perhaps loss of limb or life. Wound suppuration may lead to the sequestration of bone denuded of its coverings. In the presence of a fracture delayed or non-union is likely to follow and massive sequestration may occur.

3. The collagenous tissues, tendon, fascia, ligament, cartilage and bone cortex are vulnerable to exposure even without wound suppuration. They must be successfully covered with healthy soft parts within a few days if they are to survive. Many wounds, therefore, cannot be left wide-open as a precaution against wound suppuration without sacrifice of exposed collagenous tissues.

4. Fortunately, the mere fact that wound suppuration has occurred does not mean that tendons will be destroyed, joints are doomed to destruction, fractures will not unite and bone will be lost by sequestration. Prompt surgery including excision of all dead and devitalized tissue, effective dependent drainage, adequate immobilization and the staged closure of wounds together with the adjuvant use of antibiotics and whole blood transfusions is likely to eliminate the septic process and salvage the tissues potentially destroyed by it. Moreover, the prophylaxis of wound sepsis is the surgical eradication or prevention of the pabulum of sepsis. This concept places the major emphasis on the pathology rather than the bacteriology of a potentially septic wound.

## QUESTIONS

OSCAR P. HAMPTON, JR. (St. Louis, Mo.): The Moderator instructed us beforehand to make these talks informative and provocative. This first question I am afraid I provoked from some friend in the audience by conversation during World War II over cognac and other things, rather than by my talk. Do you believe that the use of prophylactic gas gangrene antitoxin has any merit? My answer is absolutely none. I think that the use of prophylactic gas gangrene antitoxin is a great deficiency in American surgery. I wish I had

the incentive and the ability to prove it experimentally. Certainly those authorities on the subject with whom I have discussed it are unanimous that it has no merit. It is my understanding—and I am sure I am correct—that the Armed Forces do not use it. In our theater during World War II, casualties of one regiment were given prophylactic gas gangrene antitoxin at the aid station and those of another regiment were not given it. This was during a period of time when we were having a relatively high incidence of gas gangrene. The regiment whose casualties got the antitoxin had more gas gangrene than the one which did not get it. Certainly, we should stop sensitizing people to horse serum with prophylactic gas gangrene antitoxin.

That brings to mind something that I believe is important to this Association. There is no greater need in American surgery than for a complete clarification of the status of tetanus toxoid immunization, how long it lasts, when reimmunizing doses must be given, and whether those of us who were in the Armed Forces and have not had it since 1944 can take a stimulating dose now and be immune. I am told by such authorities as Dr. Lyons that we could, but it is not practiced. It is an important thing and it will be an important thing in the event of civilian disaster.

I have been asked to comment on the tight closure of badly traumatized areas. The thing I wanted to provoke in my discussion was this: There is definitely a place for the staged closure of wounds in civilian surgery. As someone has commented in an informal session at this meeting, too many people say we débride wounds in battle surgery and then close them later, but in civilian surgery we close them primarily. There definitely is a place for the delayed closure of wounds in civilian surgery. The question was on the tight closure of traumatized areas when hemostasis cannot be obtained. The objective of surgery of a wound is the elimination of the pabulum of sepsis. That means the devitalized tissue and the dead space with blood clot. It is contaminated. We can never be certain that by débridement we have completely eliminated all of this from a wound, particularly in an open fracture and particularly in the thigh. The greatest evidence of an adequately débrided wound is a clinically clean wound four or five days later. I am not sure who coined that expression, but I give Champ Lyons credit for it, as he expounded many of the principles which I have been talking about today during World War II.

The question of the tight closure of a traumatized area when hemostasis cannot be obtained seems to set up that phase of civilian surgery in the management of wounds wherein débridement is accomplished, followed by an open wound for four or five days and then, if the wound is clinically clean, a delayed closure. The tissues which are vulnerable to exposure will not slough out in four or five days. Cortical bone, articular cartilage, fascia and tendon can be covered within four or five days, when we know the wound is clinically clean, and those tissues will be salvaged. They will not die during that interval.

I have been asked what place I think antibiotics have in the prophylaxis of infection in clean and contaminated wounds. I will begin by stating that instead of being given as prophylaxis against infection, antibiotics are given against impending infection. The use of them with a clean wound is to counteract those bacteria capable of invasive infection of living tissue which may have gone into the wound from somebody's nose or throat. There is a place for it, in my opinion, in the open wound which has remained open and which may have had hemolytic streptococci breathed into it. It is to prevent invasive infection. It will not prevent the septic breakdown of dead tissue in the wound.

# PRIORITY EVALUATION AND MANAGEMENT OF MULTIPLE INJURIES*

FRANK GLENN, M.D.
*New York, New York*

THE priority evaluation and management of multiple injuries is a very broad subject. It is one that cannot be treated briefly. I therefore invite your attention to the consideration of three main points: The first is the preliminary measures that should be employed generally in the presence of trauma; second is to take up one of the most crucial groups, the unconscious patient; and third, to evaluate the injuries and the priority that should be granted them.

The American Automobile Association estimated that more than 35 million cars would be on the highways during a recent holiday weekend. They predicted as well that the three-day period would see over 300 deaths due to traffic accidents. Actual reports came to almost 400 but they did not take into account victims who died later as a result of the injuries they incurred that weekend. Between 32,000 and 35,000 deaths in the United States each year are estimated to be due to automobile accidents. It is the responsibility of the medical profession to save as many of these casualties as possible.

As the network of roads across this great country is extended and the number of motor vehicles using the highways increases, a higher incidence of accidents must be anticipated. Improvements in highway construction, closer inspection of cars and drivers, and more rigid enforcement of traffic regulations are the obvious means of keeping accidents at a minimum. However, since successful prevention of accidents depends in no small part on the public's response to attempts at control, it is likely that the number of accidents will increase before the controls are effective. The mortality rate and the extent of disability from automobile accidents are high for several reasons:

1. The type and extent of the injuries themselves. Their magnitude tends to be in direct proportion to the speed of the vehicles involved. The speed now reached on some highways is high but it will probably go higher. Many of these injuries are instantly fatal and little can be done about them.

2. The practical difficulty of rendering the injured the immediate, simple measures that can save life such as relief of an obstructed airway or control of massive hemorrhage.

3. Delay in transporting the injured from the scene of the accident to where definitive treatment may be initiated. Time and distance are definite factors.

4. The variation in management and care accorded patients after their arrival at the hospital.

Any mortality rate that is the result of accidents which could be avoided must be looked upon as excessively high. If hospital clinical records are reviewed in conjunction with the postmortem findings, it becomes evident that some of these patients might have been saved. Although some of the factors involved are lacking, such studies direct attention to the contention that often the injuries themselves need not have been fatal. This conclusion appears justified particularly in cases of multiple injuries which are so common in automobile accidents. Based on the same studies, it follows that the treatment of single injuries has been more satisfactory than that accorded multiple injuries. It is furthermore suggested that when multiple injuries occur, they are frequently not recognized and are improperly evaluated. Particular attention should be directed, therefore, to improving the care accorded all highway casualties. Indeed, their examination and care are in need of definite revision.

Evaluation of patients with multiple injuries should include not only an estimation of the various wounds but also of the degree of shock, the extent of hemorrhage and the general status of the patient as to age, state of nutrition and coexisting systemic diseases, if any. Recognition of multiple injuries indicates that added caution is essential. The coexistence of

* From the Department of Surgery, The New York Hospital-Cornell Medical Center, New York, N. Y.

two injuries in the same patient represents a greater hazard and increment than do trauma and injuries which occur separately. When this situation is increased to triple or quadruple injuries with their consequent greater contribution to shock, we are faced with one of the most difficult problems in care of the injured.

Time is always important. Marked deterioration of the body's economy as the result of wounds may occur within a very short period, yet with little outward evidence of change. Unmolested, an injured person may appear to change very little over an hour or two, yet when disturbed for extensive examination or treatment sudden and irreversible shock may be precipitated.

Triage, the sorting and evaluation of the wounded, as developed in both World Wars, is well established. In time of war the chief problem is how best to care for a large number of casualties in a comparatively short space of time, in what is usually a small area, with limited equipment and personnel. With injuries that occur on our highways the problem is somewhat reversed. The casualties are comparatively few in number and apt to be distributed over a wide area too often at some distance from available medical care. In lapse of time the actual distance between the scene of an accident and hospital facilities is rapidly diminishing. Transportation is becoming easier. The interval between injury and primary therapy grows constantly shorter. However, the steady rise in the number of accidents is placing a greater responsibility on the hospitals and their facilities. Thus all hospitals throughout the country should be alert, not to an influx of injured at any one time but to giving a few individuals each year the meticulous care they require. The problem is one of evaluating these patients and determining the priority of treatment needed for their various injuries when they are multiple. It is important, therefore, that a simple, practical plan based on sound principles of therapy be evolved which will enable those who find themselves responsible for the care of the injured from the highways to salvage as many of these casualties as possible.

Better results with severely traumatized patients will be possible if a specific routine is undertaken promptly at the hospital. Preservation of life, conservation of tissue and maintenance of function are the three chief objectives to be kept in mind. Throughout the course of care and treatment the patient needs repeated evaluation so that nothing will be overlooked. He is first a problem in survival, second of conserving parts, and third restoration of function.

Hospital facilities and availability of transportation have so increased in the past two decades that a much greater percentage of those who are injured on the highways now reach a hospital alive. It is at the hospital, wherever it may be, that a desirable type of routine should be employed. Because of the large number of hospitals in this country the task of establishing a routine that is safe, efficient and applicable in all cases is rather difficult. Perhaps much could be accomplished if specific instructions, printed in large type, were posted in the admitting and emergency rooms of all hospitals. These instructions should deal with the fundamental concepts of care for the injured and should emphasize not only what should be done but also what should not be done. They should include specific suggestions and might be listed as follows:

1. Adjust the patient's position (*a*) to give him an adequate airway, (*b*) to compensate for blood loss and (*c*) to prevent aspiration of vomitus.

2. Remove all clothing and inspect the entire body surface before anything else is attempted.

3. Make a complete and thorough physical examination so that no injury can be overlooked and at the same time establish a baseline for comparing changes that may follow.

4. Make only the simplest adjustments when necessary and use only temporary measures on injured parts, i.e., additional dressings to large wounds, temporary splints and position support.

5. Conserve body heat with light blankets or such measures as may be available, both during and after evaluation.

6. Administer fluids to prevent or combat shock. Dextrose in saline should be started immediately, followed by plasma as soon as it is available and finally by whole blood when cross matching has been completed. The lapse of time before administration of whole blood should be as short as possible. In most hospitals this can be accomplished within two or three hours.

7. Special examinations, especially x-ray, should be done with the least possible disturb-

ance to the patient. For patients with multiple injuries, portable x-ray equipment may be preferable to shifting the patient from stretcher to fluoroscopic table, but this must be left to the physician in charge. Improved x-ray facilities now permit plates to be taken or fluoroscopy carried out with very little further trauma to the patient.

8. During the period of examination, when blood pressure levels are likely to be low, oxygen therapy will prove beneficial in many cases. The use of vasocontractors to elevate blood pressure during an examination usually results in further blood loss or increased shock.

9. Nothing should be given by mouth.

10. An indwelling urethral catheter will furnish additional information on the genitourinary tract immediately as well as provide a constant record of urinary output.

These ten steps cover the essential points of evaluation and at the same time provide for initial and proper care of the seriously injured. While none of them should be considered as dealing with definitive treatment, they may be life-saving by preventing incidents that might be fatal, such as obstruction of the airway, continuation of hemorrhage or early development of irreversible shock.

Our next concern is to catalogue the different types of wounds and their locations according to their relative importance as to priority for definitive treatment. Generally speaking, bleeding vessels or hemorrhage require immediate control by pressure or by direct ligation. Obstructed airways lead to asphyxiation. Chest injuries may lead to pneumothorax. Both require that a free air passageway be restored and maintained. This may entail correction of a chest wall defect supplemented by the use of increased oxygen intake. Extravasation of urine in pelvic injuries is devastating to the patient with multiple injuries. Where this may be corrected by insertion of a catheter, nothing more is required. If, however, a catheter cannot be introduced or is not adequate, cystotomy must be done to divert the urine and drain the periurethral structures involved. Skeletal injuries require immobilization and fixation in order to prevent further shock. Intra-abdominal wounds, particularly those involving massive hemorrhage, require early operation, but with large amounts of blood available before any procedure is undertaken.

These suggestions are based on general situations which would be quickly understood and fully appreciated by any physician. More specific consideration follows on the states and phases of trauma associated with multiple injuries.

## UNCONSCIOUSNESS

Nothing so obscures complete and accurate evaluation of a severely injured person as does unconsciousness. Its existence alone is an indication of the critical state of the patient and its persistence is forboding indeed. Its mode of onset and its place in the sequence of events following the accident constitute significant information. The unconscious patient with multiple injuries requires specific evaluation on admission as to (1) blood pressure and blood volume relationships, (2) intracranial brain tissue damage and (3) consideration of previous medication, drugs or alcohol intake.

In an unconscious person with a head injury particular precaution should be taken that the airway does not become obstructed by prolapse of the tongue or regurgitation of vomitus, or by accumulation of material from the respiratory tree. Since these patients may be in shock, extreme lowering of the head may increase the intracranial pressure and cause further depression of the brain stem centers. Unless contraindicated, patients should be turned on their side with only a slight lowering of the head of the stretcher as this will place them in the safest position as far as aspiration is concerned.

Evacuation of the gastric contents and maintenance of decompression by an indwelling gastric tube should be done soon after such a patient has been seen. Because they cannot always be controlled, restraint is sometimes required with these patients; but great care should be exercised to prevent further trauma of other parts when a patient has a brain injury and is in a disturbed state. This type of patient requires continual re-evaluation of the vital signs that may indicate increasing intracranial pressure or an extension of intracranial hemorrhage. Frequent observations should be made and recorded on pulse, blood pressure and respiration rate. Repeated testing of reflexes and response to stimuli aid in locating the brain injury.

Unconsciousness from drugs or alcohol requires that these substances be neutralized by

any means available. Gastric lavage is useful if the lapse of time since intake has not been too great. Counteracting drugs are hazardous and should be used only in unusual circumstances. If the effect of the drugs is so severe as to be considered toxic, definitive treatment should be postponed.

All patients with injuries are candidates for shock. Death can follow in the wake of shock. Vigilant and wise care is needed to prevent its development or to reverse it once it is established. In general, shock is associated with a decreased circulating blood volume. While it is unusual to find this in the early period following injury, unless due to blood loss through hemorrhage, it is common to have shock develop later from a diminishing blood volume that is the result of plasma escaping into the extravascular tissues.

The most common manifestations of shock are a rising pulse rate and falling blood pressure. These two criteria should be obtained and recorded as soon as an injured person is brought to an admitting room. They will serve as a baseline and guide during the ensuing period.

Usually gross hemorrhage is not difficult to control, but it is difficult indeed to control plasma that is escaping into the extravascular tissues, which often occurs in cases of crush injury, burns or acute pain. Sometimes it can be reversed only by replacement therapy. The role of pain is not clearly understood, but it is well known that continued pain or increased pain that is the result of manipulation can produce a state of shock. These simple facts should be kept in mind during the examination and evaluation of a patient with multiple injuries.

Because shock is so difficult to reverse once it has set in, it should be prevented if at all possible. This means prompt control of any hemorrhaging, interruption of pain, maintenance of normal body temperature and an adequate oxygen supply. Such measures will render subsequent treatment much easier. After preliminary examination intravenous administration of electrolyte solutions may prevent a trend toward shock. If shock is already present to a marked degree, these solutions can be used to support the circulation for a brief period. Thereafter plasma should be employed until whole blood is available. When shock is profound, it can seldom be corrected without administration of sufficient blood to restore the blood volume close to its normal level. Prevention of hemorrhage from a large vessel, no matter where in the body, may be impossible without an operative procedure. This is particularly true when bleeding from a great vessel begins afresh as soon as the blood pressure returns to normal. Patients who have not had acute blood loss will usually recover from a state of shock simply through the use of plasma, careful maintenance of body temperature and time. Once again a period of stabilization of an hour or more should be allowed before undertaking a corrective operative procedure.

In all cases of injury from accident one must be alert to certain conditions which may simulate shock, for they are encountered quite often. Such conditions include cardiac disease, pulmonary embolism, poisoning from alcohol, morphine, the barbituates or occasionally insulin. Not infrequently patients are brought in from automobile or other accidents suspected of internal and extensive trauma but without external evidence of injury and without a definite history of injury. To treat such patients for shock may prove fatal.

The coexistence of shock and unconsciousness, which is often found in the presence of head injuries, requires careful general evaluation before a decision is made as to primary definitive therapy. If there is no evidence of injury elsewhere, the unconscious patient in a state of shock should be considered primarily as a case of head injury and subjected to whatever procedure is most likely to correct this situation. Such procedures may be undertaken without anesthesia and will frequently result in immediate return to consciousness, but may be followed by shock to a greater degree. Because of this, measures to combat shock should be instituted and maintained as the operative procedure is carried out.

Shock that is found to be associated with chest injuries is usually the result of decreased oxygen intake, blood loss and pain. Priority should be given to restoration of adequate oxygen intake before controlling the other two situations. Maintenance of an adequate airway is of greater importance than a high oxygen concentration. The position of the trachea, which is often distorted in cases of maxillary facial injury, may require some sort of splinting such as a tube or metal airway. Failing this, tracheotomy may be necessary.

Massive intrathoracic hemorrhage arising

from vessels about the thoracic cage may be controlled readily at operation, but hemorrhage from the major branches of the aorta is far more difficult to control. Nevertheless heroic attempts at times are indicated while the patient is in a state of shock. Therefore injury to the larger systemic vessels may warrant operation even though the patient has not recovered from shock. Patients with intra-abdominal injuries who are in a state of shock should be allowed time for correction of the shock before attention is given to the injuries. This may require administration of large quantities of blood to re-establish the blood pressure and the abdomen may fill from further hemorrhage, but revival from shock and a period of stabilization will offer more assurance of recovery than a procedure undertaken while the blood pressure is low. Soiling of the peritoneal cavity by gastrointestinal contents or by blood undoutedly contributes to shock, but its rate of damage is usually slower than the rate of recovery from shock which can be accomplished by the use of whole blood. Decompression of the stomach, rectum and bladder with indwelling tubes will aid in maintaining shock at a minimum.

No rule of thumb can be applied to the treatment of multiple injuries for there will be exceptions to any general rules established. Nevertheless, wounds that tend to deplete the body economy of any element essential to life merit primary attention to say the least. For example, one of the basic demands of the body is oxygen. Any injury which interferes with the body's acquisition of this need will very quickly result in death. The seriousness of blood loss is proportionate to the rate of hemorrhage. But to a certain extent the vascular system enjoys a better protective mechanism than does the respiratory tract, since the rate of blood loss diminishes with the fall in blood pressure, except in the very large vessels. Other variations may be found in the gastrointestinal tract. Perforation calls for correction but less urgently since a longer lapse of time between injury and definitive treatment is compatible with the preservation of life.

Let us consider a patient who has sustained an injury in each of the major divisions of the body, all at one time. We will include a depressed skull fracture, fracture of the mandible, an open sucking chest wound, a penetrating wound in the abdomen, a fractured pelvis with a tear in the membraneous urethra and a fractured femur. This patient has hemorrhaged and is in shock, rendering an adequate supply of air to the respiratory system imperative, for the tissues must have oxygen. The impaired airway that is the result of the fractured mandible must be cleared at once and then a high oxygen atmosphere supplied. Further correction of this situation is next achieved by re-establishing the intrathoracic pressure with closure of the chest wound. These first measures may have to be undertaken even though the patient is still in shock. Once accomplished, an hour or two for stabilization, with continuation of supportive measures, should see the patient's condition sufficiently improved to permit the next major step.

The head injury is causing increased intracranial pressure and requires decompression proportionate to its rate of increase. In the ordinary course of events an operative procedure on the head might well take precedence over other injuries; but if there is no evidence of increasing pressure, attention should be directed first to the pelvis and the urethra. Prevention of further extravasation of urine can be accomplished by cystotomy and establishment of adequate diversion and drainage. In any event, both these conditions, the increasing intracranial pressure and extravasation of urine from the urethra, rate high priority as to early surgical correction when one is dealing with multiple injuries. Both wounds can be truly mortal if left unattended but their respective prognosis is improved by early surgical therapy.

The intra-abdominal injuries may be so varied in nature and extent as to warrant high priority. For example, a ruptured spleen may cause great and rapid loss of blood and will lead to complete exsanguination and death if not controlled by direct operation. Other injuries should be given definitive care as soon as possible but they rarely take precedence over the aforementioned.

Intra-abdominal injuries are often difficult to identify as to their exact location and extent. When viscera have been disturbed by extensive injury, an adequate supply of blood should be in reserve before operation is undertaken. Soiling of the peritoneum from bile and gastric contents is a devastating process that plays an important role in shock. This should be kept in mind when dealing with abdominal wounds.

It justifies an attack on these injuries as soon as the general condition of the patient permits.

In this hypothetical patient the injuries to the extremities and the extensive soft tissue damage should be dealt with last. It should be stressed that deliberate postponement of definitive treatment for the extremities does not constitute disregard or neglect. Prevention of further injury by splinting and fixation as well as control of hemorrhage should be considered emergency and temporary measures, but they are important in preventing further shock and deterioration of the patient's general condition.

Perhaps the most pronounced reaction of any organ to trauma is that of the kidneys. Reduction of renal function and consequent inadequate excretion of toxic substances may result from the breakdown of injured tissue. At the same time there is often retention of sodium which further contributes to a vicious circle, since impaired renal function thwarts the recovery process from trauma. The longer a patient remains in shock and untreated or inadequately treated, the greater are his chances of developing acute renal failure. Patients with multiple injuries associated with hemorrhage, pain and tissue destruction frequently experience decrease in renal function when in some degree of shock. If common preanesthetic medications or anesthesia itself are also imposed on the patient under these circumstances without a preliminary period of stabilization to reverse the trend to shock, renal function may fail completely.

Without going into greater detail, the importance of renal function to recovery and the various mechanisms that can contribute to its derangement should be stressed. They should not be discounted in the presence of trauma. Indeed, the state of water and salt metabolism under the conditions that commonly prevail after massive trauma is too often lost sight of and can lead to errors in supportive therapy that may prove fatal.

## SUMMARY

In general, a direct relationship has been found to exist between the number of traffic accidents that occur in a certain area and the density of that area's population. The greatest number of accidents occur within a reasonable distance from well equipped hospitals and medical centers. Many do, of course, take place in the less populated districts and may be far removed from any hospital facilities. Availability of good hospital care plays an important part in saving these victims, yet it seems that the mere proximity of a well equipped hospital does not always enhance the patient's chances of survival.

Any review of available records, particularly coroners' reports and their preceding clinical histories, suggests that the mortality rate from accidents on our highways could be reduced, especially with cases of multiple injury. The following precautions and suggestions are recommended in an effort to attain this goal:

1. A thorough evaluation should be made of each patient, based on a complete examination that overlooks no obscure injury.
2. There must be adherence to a prescribed routine which includes preliminary therapeutic measures in addition to evaluation.
3. To anticipate and prevent, or recognize and treat shock is of primary importance.
4. Unconsciousness is in itself indicative of a critical situation that requires added caution in evaluation and treatment.
5. A realization that improper care is no better than neglect emphasizes the need for caution in situations that are ill defined or controversial.
6. No absolute rule of thumb should be followed in the management of a patient with multiple injuries, but those wounds which tend to deprive the body of any element that is essential to life should be treated first.
7. Responsibility for the continuity of a patient's over-all care should be invested in one individual only.

## QUESTIONS

FRANK GLENN (New York, N. Y.): A good question which comes up with our house staff quite frequently is what should be done with the patient who appears to be intoxicated as well as severely injured, whether or not unconscious. I believe, in general, that gastric lavage accomplishes two things: It removes the undigested food and tends to diminish the likelihood of aspiration, and it removes such alcohol as may remain to be absorbed. Then you must watch your patient and wait as long as you can before embarking on any decisive therapy. There are so many variables to this that I can say little more than I think gastric lavage is most important.

What are the time and indications for tracheotomy in crushed chest or multiple rib fractures?

Tracheotomy is being done, quite properly, earlier today than it was a few years ago. In the crush injury of the chest it is well to bear in mind that you may have small hemorrhages within the parenchyma of the lung, draining into the respiratory tree. In the absence of a hemothorax, namely, if you have fractured ribs but no decrease from hemorrhage in the apparent area, I believe that you can usually take care of those patients very well with simple measures and sometimes gastric decompression. Failing this and in the presence of continued cyanosis, after directing attention to position and aspiration, tracheotomy can be done. In tracheotomized patients you can facilitate the removal of the accumulating material in the respiratory tree by suction.

Which takes priority? (*a*) Chest, tension pneumothorax, or sucking wound? Of course both can be very devastating. I would say the sucking wound will probably get you into trouble more quickly.

(*b*) Abdomen, ruptured viscus? (*c*) Associated fractures? I take it that this individual really has two questions. Certainly the chest takes priority over the other two. If there is a ruptured viscus that is associated with hemorrhage, it seems to me that the abdomen takes priority over the fracture. If it is a ruptured viscus with intestinal contents escaping into the peritoneal cavity, it should be dealt with first. If the fracture is an open fracture and associated with hemorrhage, I think probably it should be dealt with first.

Do you concurrently splint and employ traction in associated fractures? I would answer that in this way: You are after immobilization and protection from further trauma. Therefore I believe that you should employ whichever method is the best for that particular fracture in order to accomplish immobilization of the fractured part.

Does associated craniocerebral injury contraindicate the beginning of definitive management for a fractured femur? If the patient's craniocerebral injury is not associated with a deteriorating process, if he is stabilized and if he is improving, I think you can take care of the fractured femur.

Please mention good transportation to and in the hospital. Good transportation means immobilization and protection of your patient. Therefore, put the patient in the safest position and use all means to prevent disturbance of the parts.

# THE AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA

## *OFFICERS*

**1952–1953**

***President:*** Martin C. Lindem, M.D., *Salt Lake City, Utah*

***President-Elect:*** Eslie Asbury, M.D., *Cincinnati, Ohio*

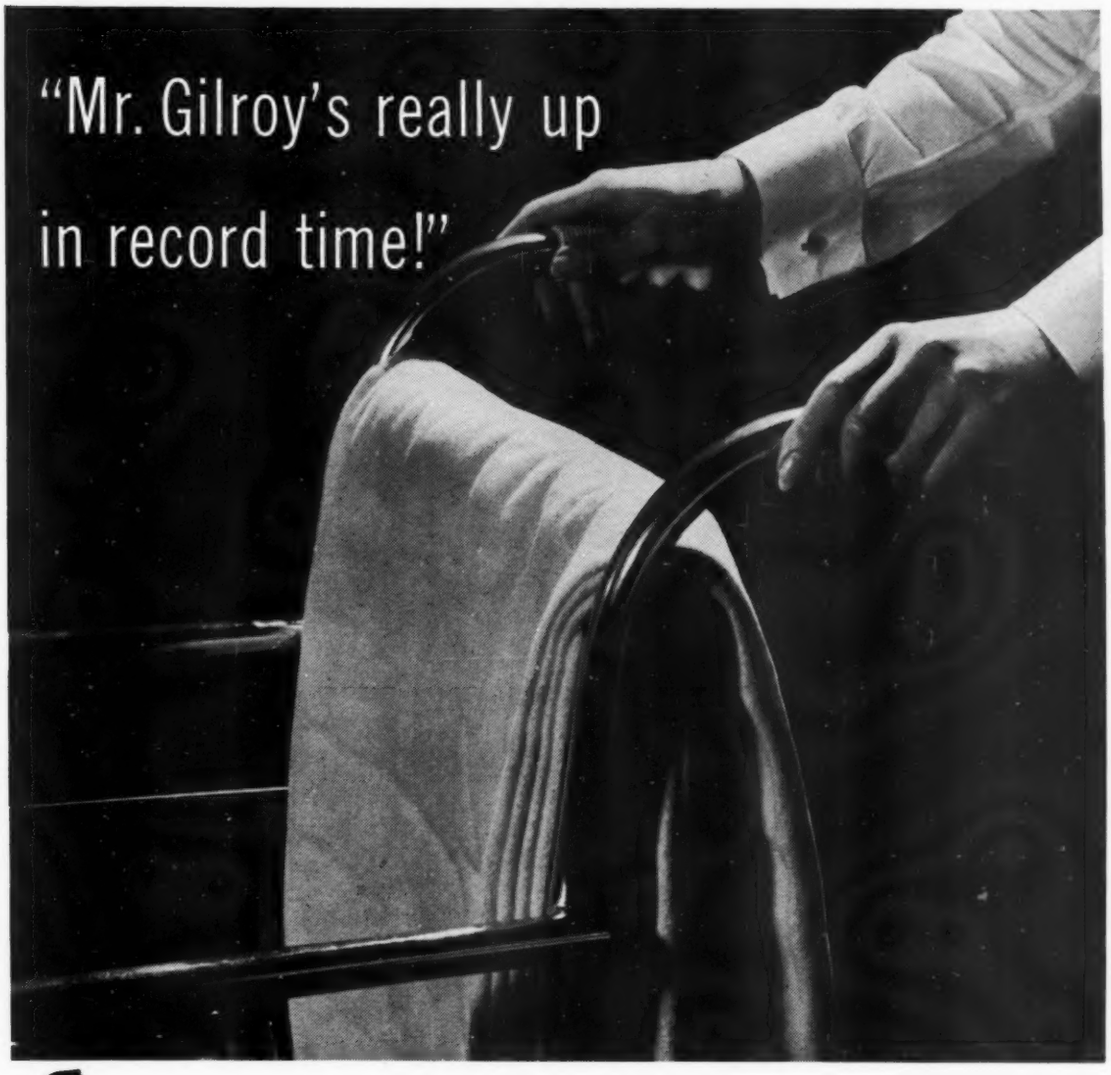
***Vice-President:*** Robert H. Kennedy, M.D., *New York, N. Y.*

***Secretary:*** James K. Stack, M.D., *Chicago, Ill.*

***Treasurer:*** Raymond Householder, M.D., *Chicago, Ill.*

***Recorder:*** Harrison L. McLaughlin, M.D., *New York, N. Y.*

....INSTEAD OF UNPHYSIOLOGICAL "PHYSIOLOGICAL SALINE"*




## Here's how new POLYSAL†/Cutter helps your patients:

**1. POLYSAL** prevents and corrects hypopotassemia without danger of toxicity.[1]

**2. POLYSAL** corrects moderate acidosis without inducing alkalosis.[1]

**3. POLYSAL** replaces the electrolytes in extracellular fluid.[1]

**4. POLYSAL** induces copious excretion of urine and salt.[1]

Polysal, a *single* solution to build electrolyte balance, is recommended for electrolyte and fluid replacement in all medical, surgical and pediatric patients where saline or other electrolyte solutions would ordinarily be given. Write for literature and handy wallet-size mEq chart . . . Cutter Laboratories, Berkeley, California.

1. Fox, C. L. Jr., et al.: An Electrolyte Solution Approximating Plasma Concentrations with Increased Potassium for Routine Fluid and Electrolyte Replacement, J. A. M. A., March 8, 1952.

†*Cutter Trade Mark*

*In distilled water—*
*250 cc. and 1000 cc.*

•

*In 5% Dextrose—*
*500 cc. and 1000 cc.*

CUTTER

## *MAKE *New* POLYSAL YOUR ROUTINE PRESCRIPTION

# THE NEW YORK POLYCLINIC
## MEDICAL SCHOOL AND HOSPITAL
**(ORGANIZED 1881)**

***(The Pioneer Post-Graduate Medical Institution in America)***

### SURGERY and ALLIED SUBJECTS

A combined surgical course comprising general surgery, traumatic surgery, abdominal surgery, gastroenterology, proctology, gynecological surgery, urological surgery. Attendance at lectures, witnessing operations, examination of patients pre-operatively and post-operatively and follow-up in the wards post-operatively. Pathology, radiology, physical medicine, anesthesia. Cadaver demonstrations in surgical anatomy, thoracic surgery, proctology, orthopedics. Operative surgery and operative gynecology on the cadaver; attendance at departments and general conferences.

### RADIOLOGY

A comprehensive review of the physics and higher mathematics involved, film interpretation, all standard general roentgen diagnostic procedures, methods of application and doses of radiation therapy, both x-ray and radium, standard and special fluoroscopic procedures. A review of dermatological lesions and tumors susceptible to roentgen therapy is given, together with methods and dosage calculation of treatments. Special attention is given to the newer diagnostic methods associated with the employment of contrast media, such as bronchography with Lipiodol, uterosalpingography, visualization of cardiac chambers, peri-renal insufflation and myelography. Discussions covering roentgen departmental management are also included.

### EYE, EAR, NOSE AND THROAT

A three months' combined full time refresher course consisting of attendance at clinics, witnessing operations, lectures, demonstration of cases and cadaver demonstrations; operative eye, ear, nose and throat on the cadaver; clinical and cadaver demonstrations in bronchoscopy, laryngeal surgery and surgery for facial palsy; refraction; radiology; pathology, bacteriology and embryology; physiology; neuroanatomy; anesthesia; physical medicine; allergy; examination of patients preoperatively and follow-up post-operatively in the wards and clinics; attendance at departmental and general conferences.

### FOR THE GENERAL PRACTITIONER

Intensive full time instruction covering those subjects which are of particular interest to the physician in general practice. Fundamentals of the various medical and surgical specialties designed as a practical review of established procedures and recent advances in medicine and surgery. Subjects related to general medicine are covered and the surgical departments participate in giving fundamental instruction in their specialties. Pathology and radiology are included. The class is expected to attend departmental and general conferences.

**FOR INFORMATION ABOUT THESE AND OTHER COURSES ADDRESS**

**THE DEAN, 345 West 50th Street, NEW YORK 19, N. Y.**



# COOK COUNTY
# GRADUATE SCHOOL OF MEDICINE

***Postgraduate Courses*—1953**

SURGERY—Intensive Course in Surgical Technic, Two Weeks, starting March 16, March 30, April 13
Surgical Technic, Surgical Anatomy & Clinical Surgery, Four Weeks, starting June 1
Surgical Anatomy & Clinical Surgery, Two Weeks, starting March 16, June 15
Basic Principles in General Surgery, Two Weeks, starting March 30
Gallbladder Surgery, Ten Hours, starting April 20
Surgery of Colon & Rectum, One Week, starting April 13
General Surgery, One Week, starting May 4
General Surgery, Two Weeks, starting April 20
Fractures & Traumatic Surgery, Two Weeks, starting June 15

GYNECOLOGY—Intensive Course, Two Weeks, starting March 16
Vaginal Approach to Pelvic Surgery, One Week, starting March 30

OBSTETRICS—Intensive Course, Two Weeks, starting March 30

PEDIATRICS—Intensive Course, Two Weeks, starting April 6
Congenital Heart Disease, Two Weeks, starting May 18

MEDICINE—Intensive General Course, Two Weeks, starting May 4
Electrocardiography & Heart Disease, Two Weeks, starting March 16
Allergy, One Month and Six Months, by appointment

UROLOGY—Intensive Course, Two Weeks, starting April 13
Ten-Day Practical Course in Cystoscopy starting every two weeks

DERMATOLOGY—Intensive Course, Two Weeks, starting May 11

**TEACHING FACULTY—ATTENDING STAFF**
**of Cook County Hospital**

Address Registrar, **707 South Wood Street, Chicago 12, Illinois**

# Precision
## FOR INCISION

As the "Master Blade" for the Master Hand, where the need is for PRECISION, every Crescent Blade is precision-made for fine balance . . . precision-honed for extreme sharpness . . . precision-tested for strength and rigidity.

Precision-performance is assured by the new Swedish steel of high carbon content and unusually fine grain.

Precision-protection is provided by the new moisture-proof, all-climate, aluminum-foil wrapping.

*Samples on request*

CRESCENT SURGICAL SALES CO., INC. • 440 4th Ave., New York 16

Crescent

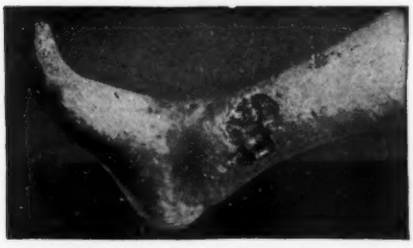
SURGICAL BLADES AND HANDLES

*for the successful treatment of...*

## LEG ULCERS

**the DAXALAN-DOME-PASTE BANDAGE TECHNIQUE**

AS EMPLOYED in the Department of Peripheral Vascular Diseases—New York Polyclinic Medical School and Hospital.



DOMEBORO TABS is listed as "BUROW'S SOLUTION-DOMEBORO TABS", on Page 376, in the "Manual of Dermatology"—issued under the auspices of the National Research Council and is recommended to be used wherever Burow's Solution is indicated.

This technique is based on a 3 point program:

1. Reduce the dermatitis with wet dressings of DOMEBORO TABS (BUROW'S SOLUTION).
2. Combat local infection and stimulate healing with thick application of DAXALAN in the center of the ulcer and surrounding areas.
3. Overcome venous insufficiency, statis and edema by wrapping DOME-PASTE BANDAGE (Unna's Boot) around the entire leg to supply compression.

Dome-Paste Bandage is Now Accepted by the A.M.A. Council on Physical Medicine and Rehabilitation

DOCTOR!
You may now prescribe
SOOTHING
Modernized — Simplified
Fresh and Stable
BUROW'S SOLUTION
1:20
In four forms
DOMEBORO
Tablets • Packets • Powder
Ointment
Write for samples and reprints from medical literature

**DOME CHEMICALS, INC.**
109 W. 64th STREET, NEW YORK 23, N. Y.
*Makers of the Soothing, Modernized Form of Burow's Solution*
DOMEBORO—Tablets • Powder • Packets • Ointment

*For*
*Comfort . . .*
*Neatness . .*
*For*
*Support . . .*
*Compression*

*The Depend-able Original*
**E-L-A-S-T-I-C**
***Adhesive Bandage***

# ELASTOPLAST
**Trade Mark**

Molds and holds to any contour . . . Allows joint motion . . . Provides firm support and controllable compression . . . Does not slip. Assures the neatest, most comfortable dressing in private and hospital practice. Available at your dealer in widths of 1, 2, 2½, 3, 4, 5 and 6 inches, 3 yards long (slack).

*** Elastoplast is also available in convenient unit dressings and compresses. Write for literature.**

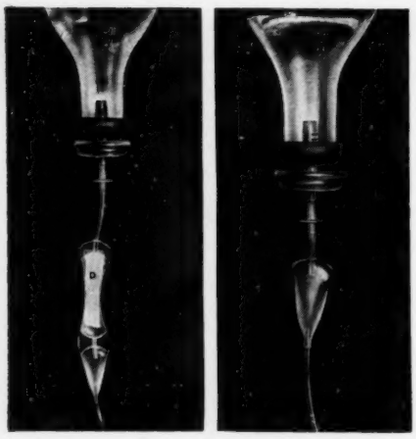
**DUKE** LABORATORIES, INC.
STAMFORD CONN.

---

*Tomorrow's technic today . . .* **Introducing the**

# TEL-O-VAC® DISPENSING SEAL

***A 3-function dispensing closure that simplifies the administration of intravenous fluids and blood, and serves as a vacuum seal in the preparation of sterile solutions.***



## Fenwal UNIVERSAL SETS

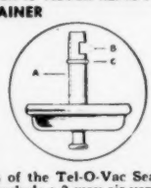
Disposable Dispensing Sets for the administration of intravenous solutions and blood. Both Fluids and Blood Sets may be used with all types of conventional closures as well as the recently devised Fenwal Blood Pack*.

**Permits Better Control of Flow**

Infusion time can be reduced by completely filling Filter Chamber (D) with blood before starting the transfusion. This is readily done by gently squeezing the plastic filter. The flexible character of both filter and drip chambers affords a means of creating most favorable conditions for steady, uninterrupted results.

The Fenwal Plastic Filter Chamber may be gently squeezed to free or break up any blood clots that may tend to clog at the outlet tube or needle.

- **NO AIR VENT TUBE IS EVER REQUIRED**
- **STOPPER IS NEVER REMOVED FROM CONTAINER**




The stem of the Tel-O-Vac Seal is fabricated to include a 2-way air vent (A) and inside strainer (B) as illustrated. Note supporting ring (C) which establishes the proper point at which the Seal should be set prior to attachment of Fenwal Universal Sets.

*Sack, Theodore et al, The Preservation of Whole ACD Blood Collected, Stored, and Transfused in Plastic Equipment, Surg. Gyn. Obst.: 95, 113-119, 1952.

Walter, Carl, W. A New Technic for Collecting, Storage and Administration of Unadulterated Whole Blood. Surgical Forum.

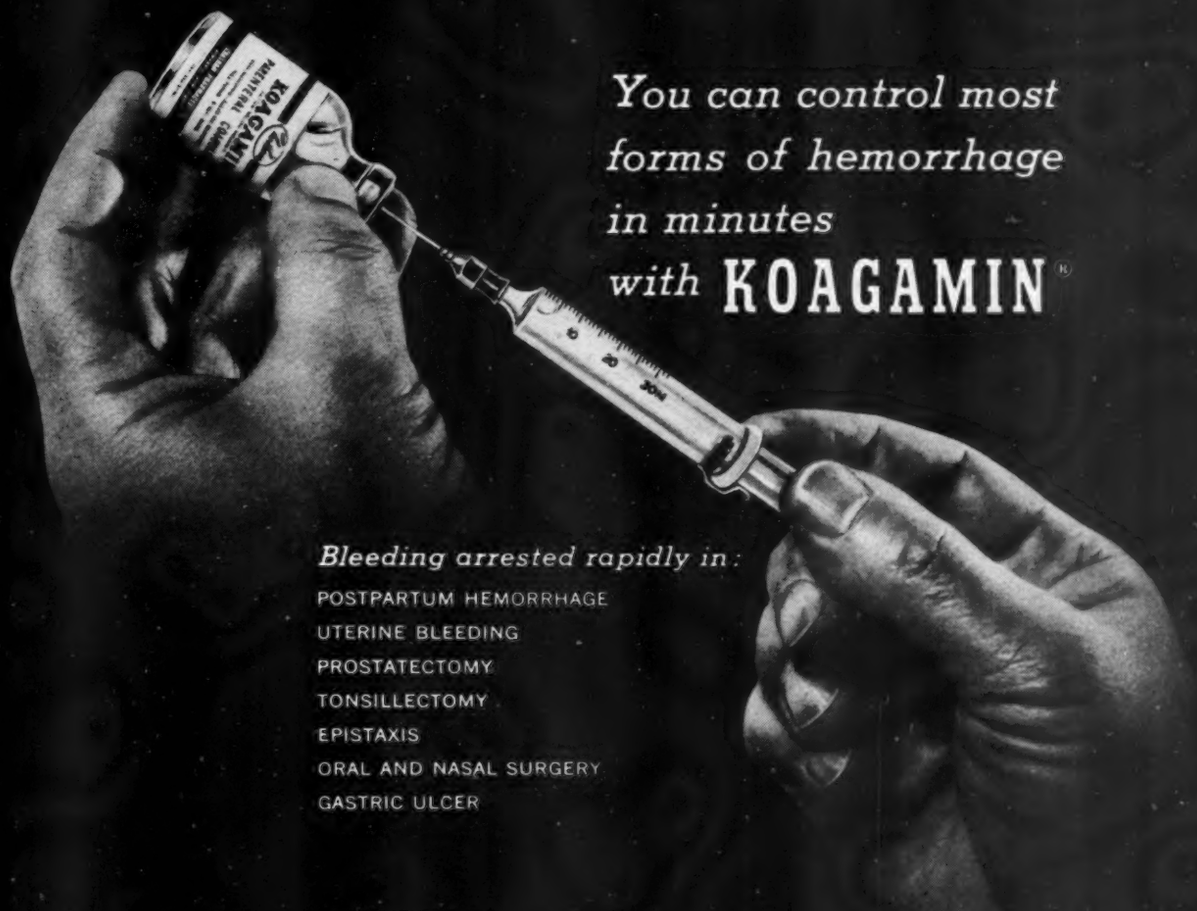
Walter, Carl W., and Murphy, Wm. P., Jr., A Closed Gravity Technic for the preservation of Whole Blood in ACD Solution utilizing Plastic Equipment. Surg. Gyn. Obst.: 94, 687, 1952.

**MACALASTER BICKNELL PARENTERAL CORP.**
**243 Broadway Cambridge 39, Massachusetts**

*Branch Offices: Atlanta, Ga. • Columbus, Ohio • Milleville, N. J. • New Haven, Conn. • New York, N. Y. • Philadelphia, Pa. • Shreveport, La. • Syracuse, N. Y. • Washington, D. C.*

*ORDER TODAY or write for further information*

THE SOLUTION DESIRED — THE Fenwal SYSTEM — AT THE INSTANT REQUIRED



*You can control most forms of hemorrhage in minutes with* **KOAGAMIN**®

*Bleeding arrested rapidly in:*

POSTPARTUM HEMORRHAGE
UTERINE BLEEDING
PROSTATECTOMY
TONSILLECTOMY
EPISTAXIS
ORAL AND NASAL SURGERY
GASTRIC ULCER

**PREOPERATIVELY** A clear field of operation, less oozing, less cauterization, less local hemostatics.

**POSTOPERATIVELY** Helps prevent secondary bleeding.

***Koagamin acts in minutes***— regardless of the cause of bleeding. Vitamin K acts slowly and is indicated only where there is prolonged prothrombin time. Where you want more rapid action use KOAGAMIN with Vitamin K.

Aqueous solution of oxalic and malonic acids for parenteral use
Supplied in 10cc. diaphragm-stoppered vials

*"Keep a vial where you can lay your hands on it."*

Chatham

*Available through Your Physician's Supply House or Pharmacist.*

**CHATHAM PHARMACEUTICALS, INC.** NEWARK 2, NEW JERSEY, U.S.A.

## WYDASE IN OFFICE PRACTICE

Part of a Series on Its Everyday Uses

Wyeth®
Philadelphia 2, Pa.

# THE MANAGEMENT OF RENAL LITHIASIS

(New use, now Council-accepted)

It is generally accepted, although not entirely established, that kidney stones may result from a change in the normal relation between colloids and crystalloids in the urine.

The colloidal state of the urine in lithiasis patients is altered by subcutaneous injections of Wydase. This results in clarification of cloudy nonpurulent urines and inhibits the formation of new crystalline material of all types by increasing their solubility.

This effect of Wydase on the urine has been found useful in the management of:

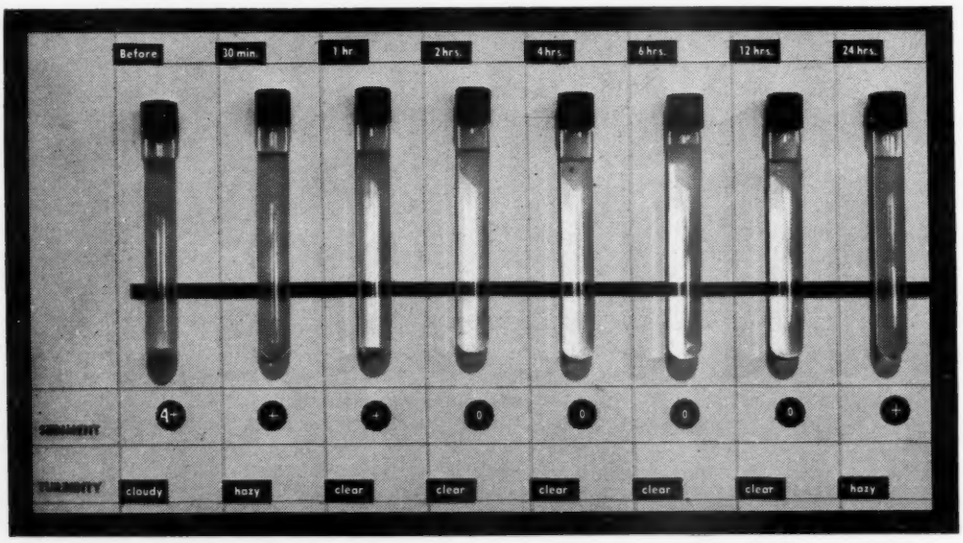
Multiple, rapidly recurring small calyceal stones

Renal surgery requiring drainage

Any calculous condition which presents surgical hazards

The Council on Pharmacy and Chemistry of the A.M.A. has now accepted the use of Wydase in management of renal lithiasis.

WYDASE INJECTIONS REDUCE TURBIDITY AND SEDIMENT OF URINE




Uncentrifuged specimens of urine show marked decrease in turbidity and sediment after a single subcutaneous injection of Wydase. (Note contrast in visibility of black line behind tubes.) Specimens collected at varying intervals over 24 hours. Clarification occurred one hour after injection. Maximum effects are evident at 2, 4 and 6 hours.

LYOPHILIZED

# WYDASE®

HYALURONIDASE, WYETH

—for use in renal lithiasis—is supplied in multiple dose vials of 1500 turbidity-reducing (TR) units. Also available in vials of 150 TR units.

# more rapid relief of pain WITH THE **motorized intermittent tractolator***

## WHEN CERVICAL TRACTION IS INDICATED

"Some patients who had previously received 5 to 8 pounds of constant traction without relief obtained rapid relief when 25 to 50 pounds of force were applied . . . Constant inadequate traction will not provide relief of pain; whereas adequate traction, even though applied intermittently, will often provide relief."

Intermittent Traction for Herniated Discs—by Bernard Judovich, M.D., Golda R. Nobel, M.D. Medical Times, Jan. 1952; Pain Syndromes—Bernard Judovich, William Bates. F. A. Davis Co., Philadelphia, 4th edition (in press) 1952.

### Suitable for Hospital or Office Therapy

- *Comfortable*
- *Sturdy*
- *Automatic*

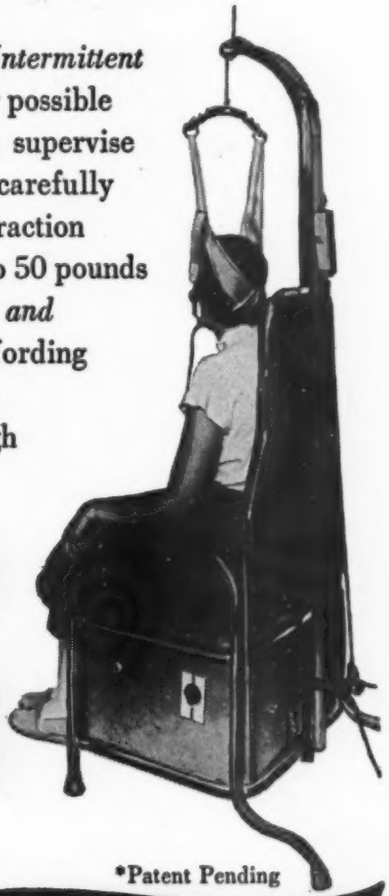
The *Motorized Intermittent Tractolator* is now in daily use in physicians' offices and hospitals. It is a dignified, handsome addition to your professional equipment, designed in consultation with the medical profession. Duration and amount of pull are automatic, once set for treatment. The apparatus is easily adjusted to the individual patient. Complete, simple instructions are provided with each Tractolator.

**The *Motorized Intermittent Tractolator* is also available as a bed apparatus for cervical and lumbar traction. Details on request.**

Pain in the Shoulder, Neck, and Arm; Brachial Plexus pain due to:

- **Herniated Cervical Disc**
- **Osteoarthritis of the Cervical Spine**
- **Narrowed Intervertebral Foramen**
- **Other Conditions in Which Cervical Traction is Indicated**

With the *Motorized Intermittent Tractolator*, it is now possible for the physician to supervise administration of a carefully graduated, smooth traction force with pulls up to 50 pounds *. . . safely, precisely and automatically* . . . affording relief of pain often unobtainable through other techniques.



*Patent Pending

**WRITE FOR INFORMATION, CASE HISTORIES, LITERATURE**

Louis Yellin, Inc.
Surgical & Orthopaedic Appliances
251 South 17th Street
(Medical Tower Building)
Philadelphia 34, Pa.

# INDISPUTABLE PROOF of an INDISPENSABLE SERVICE

# New 5TH EDITION MODERN DRUG AND THERAPEUTIC

**PUBLISHED EVERY 3 YEARS; MODERN DRUGS**

Dramatic proof of the finger tip reference value of THE MODERN DRUG ENCYCLOPEDIA and its bi-monthly supplementary service, MODERN DRUGS, comes direct from 3,000 of their 50,000 doctor and druggist users. Results of an independently* conducted reader-research effort show overwhelming dependence upon this quick reference service—complete from description to prescription for authoritative data on new ethical drugs. Com-

**THE MODERN DRUG ENCYCLOPEDIA**
is handsomely bound in red fabricoid. Contains 1500 pages, size 6" x 9¼" x 2¼". POSTPAID, $15** U.S.A.; $18 FOREIGN

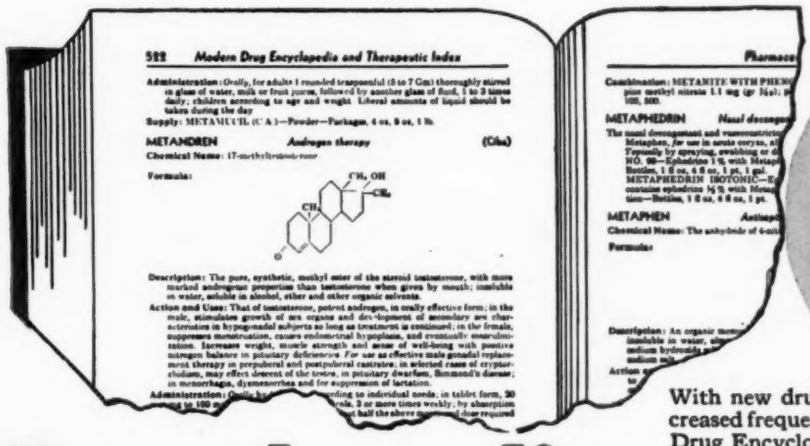
*EDITED BY*
**MARION E. HOWARD,**
M.D., F.A.C.P.
Yale University
Medical School

**MODERN DRUGS SUPPLEMENTS**

are now sent every 60 days FREE to every encyclopedia subscriber. Keeps you up-to-date between editions. Complete with cumulative index for accurate reference to all new products therapeutically and alphabetically.

## DRUG PUBLICATIONS, INC.

**49 West 45th Street, New York 36, New York**




# Encyclopedia INDEX

## SUPPLEMENTS EVERY 60 DAYS

pletely rewritten, the new 5th Edition of THE MODERN DRUG ENCYCLOPEDIA lists nearly 4,000 ethical drugs (including 1,500 brand new listings) of 175 manufacturers. Each listing includes latest composition, action, use, supply, dosage, caution and contra-indication of the drug. Here is data that you, too, will find indispensable for saving time, without sacrifice of an authoritative source.

## FINGER TIP DESCRIPTIONS AUTHORITATIVELY COMPILED IN SEVEN SPECIAL SECTIONS

- Drugs
- Biologicals
- Allergens
- General Index
- Therapeutic Index
- Manufacturer's Index

***featuring for the first time***

- Self Pronouncing Drug Listings
- Generic Name Index

* AUDIENCE ANALYSTS, Phila., Pa.

## TYPICAL COMMENTS from DOCTOR USERS

With new drugs and preparations coming with increased frequency and volume, a book such as Modern Drug Encyclopedia is indispensable.

*New York, New York*

I have owned every edition of your encyclopedia except the first. Would have bought it but did not know this book was printed. If the physicians of this country and Canada and some of the foreign countries knew its value, you would have been able to sell twice as many or more books per year.

*Memphis, Tennessee*

I like Modern Drugs because it gives honest pharmacological information; because it frequently gives contra-indications and dangers; because it describes members of a similar group in similar or identical terms. I would find it hard to practice without it.

*Green Bay, Wis.*

I have Modern Drug Encyclopedia and use the journal as supplement, thus at my finger tips I have information on new drugs.

*York, Pa.*

I don't see how a physician can know what other physicians prescribe for patients without this. It is an almost indispensable (not quite) adjunct to my practice.

*Washington, D.C.*

I find the bound book (present 5th Edition) most valuable.

*White Plains, N.Y.*

One of my most valuable books and used more often than any other that I have.

*Lincolnton, N.C.*

MAIL THIS COUPON NOW!

**DRUG PUBLICATIONS, INC.**
**49 West 45th Street, New York 36, New York**

Enclosed is the sum of fifteen dollars ($15**U.S.A.) for which please send me postpaid the new Fifth Edition of THE MODERN DRUG ENCYCLOPEDIA AND THERAPEUTIC INDEX and MODERN DRUGS. (New York City residents please add 3% for sales tax.)

NAME_____ M.D.

ADDRESS_____

CITY _____ ZONE_____ STATE_____

**Includes three-year supplementary service at $3 per year.

# Concerning VALLESTRIL* in the menopause

Vallestril is described[1] as "an effective synthetic estrogen that is singularly free from toxic side effects and complications, especially uterine bleeding. . . . Clinically, it quickly controls menopausal symptoms, . . . .

"The beneficial effect of the medication," Sturnick and Gargill continue, "appeared within three or four days in most menopausal patients. There is also evidence that the patient can be maintained in an asymptomatic state by a small daily dose, once the menopausal symptoms are controlled."

Vallestril represents the end result of an extensive series of chemical and biology studies begun in 1947. Courrier, Horeau and Jacques[2-6] found certain acid derivatives of naphthalene to be strongly estrogenic in action. Among this family of synthetic compounds, the one which showed greatest promise was the product later named Vallestril.

Vallestril proved effective orally as well as parenterally and, moreover, intrasplenic injections in castrated rats demonstrated that the drug, unlike natural estrogens, is not destroyed by the liver.

Acute and subacute toxicity studies, conducted in the Searle Research Laboratories, have demonstrated that the drug offers a wide margin of therapeutic safety.

Absence of withdrawal bleeding[1,7], in addition to avoidance of other side effects, is the safety factor which permits administration of clinically effective doses.

Vallestril (brand of methallenestril) is supplied in 3 mg. scored tablets.

*Trademark of G. D. Searle & Co.

REFERENCES:

1. Sturnick, M. I., and Gargill, S. L.: New England J. Med. *247*:829 (Nov. 27) 1952.

2. Courrier, R.; Horeau, A., and Jacques, J.: Compt. rend. Soc. biol. *141*:159 (Feb.22) 1947.

3. Courrier, R.; Horeau, A., and Jacques, J.: Compt. rend. Acad. sc. *224*:1401 (May 19) 1947.

4. Horeau, A., and Jacques, J.: Compt. rend. Acad. sc. *224*:862 (March 17) 1947.

5. Courrier, R.; Horeau, A., and Jacques, J: Compt. rend. Soc. biol. *141*:747 (July 19) 1947.

6. Jacques J., and Horeau, A.: Bull. Soc. chim. France, p. 711 (May-June) 1948.

7. Johnson, C. G.: Personal communication, April 18, 1952.

SEARLE *Research in the Service of Medicine*

Advertisement

# Abbott's COMPLETE I.V. AND BLOOD TRANSFUSING Equipment

Sterile, pyrogen-free and ready-to-use

**COLLECTING AND PRESERVING BLOOD**

For Vacuum Collection:

**ABBO-VAC®**—A-C-D Solution, U.S.P. (N.I.H. Formula B), in Universal bottles, 500- and 250-cc. sizes. Blood is drawn directly into container by vacuum. Available with sterile, disposable Blood Donor Set.

For Gravity Collection:

**NON-VAC®**—A-C-D Solution, U.S.P. (N.I.H. Formula B), in Universal bottles, 500-and 250-cc. sizes. Blood is drawn directly into container (closed technique) by gravity. Available with Donopak® 24 and 48, with or without attached, sterile, disposable needles.

**Abbott A-C-D Blood Container**—A-C-D Solution, U.S.P. (N.I.H. Formula B), in the familiar Abbo-liter® intravenous bottles, 500- and 250-cc. sizes. Blood is drawn (closed technique) directly into container by gravity. Available with Sodium Citrate 3% Solution in 500-cc. size. Also available with Donopak 24 and 48, with or without disposable needles. Designed for exclusive use with Abbott I.v. equipment.

For Storing Plasma:

**Evacuated Empty Plasma Containers**—Sterile evacuated 500- and 250-cc. Universal bottles for storing, transporting and administering plasma or serum.

**ADMINISTERING BLOOD and/or SOLUTIONS**

**Blood Recipient Set**—Sterile, disposable, ready-to-use plug-in set for administering blood from any Universal bottle or Abbo-liter type bottle. Has flexible plastic filter chamber.

**VENOPAK®**—Abbott's sterile, disposable venoclysis unit for the administration of all intravenous solutions. Converts readily to a blood recipient set with a special, disposable blood filter. For use exclusively with Abbo-liter® containers.

(Series Hookup)

**Secondary Recipient Set**—A unique, disposable unit with a built-in, flexible drip chamber and filter. Designed to plug into any Universal blood bottle and to connect with Abbott's VENOPAK dispensing cap. Allows changeover from saline to blood in a matter of moments, without removing needle from vein.

**Secondary VENOPAK**—Disposable unit designed for the continuous administration of fluids in the series hookup with VENOPAK.

**ADMINISTERING FLUIDS SUBCUTANEOUSLY**

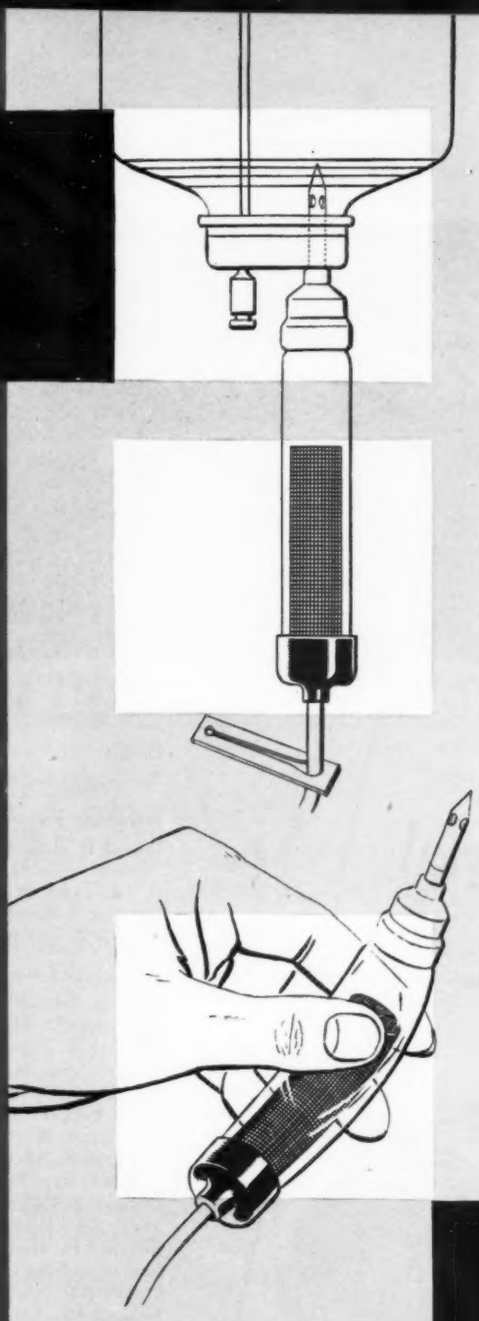
**SUB-Q-PAK®**—A completely disposable preassembled hypodermoclysis unit with plastic Y tube for administration of fluids subcutaneously.

**ADMINISTERING PENTOTHAL® SODIUM**

**VENOTUBE®**—Length of plastic tubing with attached male and female Luer adapters and pinch clamp. Allows anesthesiologist to keep syringe off the patient's arm. Pinch clamp offers additional factor of safety.

*Trade mark

1-90



**IMPROVED PLUG-IN TIP**
pre-strains blood, plasma or serum before it reaches filter. Needle-sharp canula point penetrates stopper of any universal container. No separate sterile perforating needle required.

**MONEL METAL FILTER**
permits rapid administration because of its larger filtering surface. More uniform mesh of metal filter screen assures more dependable filtration.

**FLEXIBLE PLASTIC HOUSING**
of filter chamber solves clogging problem. If canula or filter clog, a few squeezes of the non-wettable plastic housing restores free flow of blood—and in a few seconds. No need to dismantle and reassemble equipment, no need for a new venipuncture.

## Added Safeguards Increased Efficiency

WITH THE ABBOTT BLOOD RECIPIENT SET

The Abbott Blood Recipient Set is completely disposable, comes in an easy-to-store package and is sterile, pyrogen-free and ready to use. Have your Abbott representative demonstrate it. Or write us direct: Abbott Laboratories, North Chicago, Illinois. Abbott

investigate the complete **ABBOTT I. V. LINE**

# AN *Invitation* TO

## MEMBERSHIP IN A MEDICAL JOURNAL

## FOR WHICH THERE IS A DEFINITE NEED

The
American Journal
of Medicine
Not merely another Journal BUT a practical teaching Journal on Post-Graduate Medicine
PRESENTING
SEMINARS • CLINICAL and THERAPEUTIC CONFERENCES
SYMPOSIA and ORIGINAL CLINICAL INVESTIGATIONS

**Editor**
Alexander B. Gutman, N.Y.C.

**Advisory Board**
David P. Barr, N.Y.C.
Arthur L. Bloomfield, San Francisco, Calif.
Eugene A. Stead, Jr., Durham, N.C.
Joseph T. Wearn, Cleveland, O.

**Associate Editors**
Paul B. Beeson, New Haven
Herrman L. Blumgart, Boston
Eugene B. Ferris, Jr., Atlanta
Harry Gold, N.Y.C.
A. McGehee Harvey, Baltimore
George H. Houck, Palo Alto
Chester S. Keefer, Boston
William S. McCann, Rochester, N.Y.
George R. Meneely, Nashville
Walter L. Palmer, Chicago
Oswald H. Robertson, Stanford
Ephraim Shorr, N.Y.C.
DeWitt Stetten, Jr., N.Y.C.
George W. Thorn, Boston
William S. Tillett, N.Y.C.
Roy H. Turner, New Orleans
Russell M. Wilder, Bethesda, Md.
M. M. Wintrobe, Salt Lake City
W. Barry Wood, St. Louis
John B. Youmans, Nashville

THE YORKE PUBLISHING COMPANY, INC.
*also publishers of The American Journal of Surgery*

## SUBSCRIPTION ORDER FORM

THE AMERICAN JOURNAL OF MEDICINE
49 WEST 45TH STREET, NEW YORK 36, N.Y.

Please enter my subscription to the new monthly journal, THE AMERICAN JOURNAL OF MEDICINE. Subscription U.S.A. $12 per year. $13 Canada & Pan America, $15 Foreign

NAME_____ ADDRESS_____

CITY_____ STATE_____

**PROVED IN**

**THE SERVICE OF THE PROFESSION**

# GOMCO®

## EXPLOSION-PROOF SUCTION AND SUCTION-ETHER UNITS

by the thousands in hospitals all over the nation have demonstrated their ability to give you safe, convenient service.

The attractive, quiet-running unit No. 927 at right is an excellent example. A double pump model for the heaviest duty, it provides precision-regulated suction from 0" to 25" and pressure from 0 to 30 pounds. Or, for heavy-duty suction alone, specify cabinet unit No. 929, with the same quality and beauty as the "927"... both listed by Underwriters' Laboratories, Inc. and approved by CSA for use in hazardous locations, Class 1, Group C.

Ask your dealer for Gomco — the units proved in service.

**GOMCO SURGICAL MANUFACTURING CORP.**

**834-M E. Ferry Street** **Buffalo 11, N. Y.**

# Outside the Ivory Tower

THE leading national nursing organizations warned the President's Commission on the Health Needs of the Nation that nurses alone cannot solve the critical shortage of nursing service existing in almost every city and rural area in the country. The nursing groups jointly asked the President's Commission to give attention and recognition to three broad, urgent needs: (1) More nurses, through support and promotion of: recruiting to get qualified people to enter advanced, basic professional and practical nursing education programs; economic security for nurses to attract and keep workers in nursing. (2) Better nurse power, through support and promotion of improvement of schools by measures to provide for better curriculum, faculty preparation, and material resources; state legislation which will provide for the licensure of qualified professional and practical nurses in order to protect the public and facilitate interstate movement of licensed nurses. (3) More effective use of nurses through support and promotion of: better administrative practices in nursing service; elimination of discrimination in the selection and terms of employment of nurses because of race, color, or creed; improved distribution of nurses and organized nursing services in the community.

MODERN medicine is entering a new era with the development of "comprehensive" medical care of the total individual, according to Lester J. Evans, executive associate of the Commonwealth Fund. Dr. Evans compares the importance of the next twenty years in medicine with the period of 1890 to 1910 which saw the beginning of the dramatic development of scientific medicine, as physicians, teachers and researchers combined forces to solve urgent and complex problems of disease.

With the significant scientific advances of the past decades has come the realization that human health as well as human illness must be understood. Viewing medicine in a broader perspective, medical leaders of today are turning to the social and behavioral sciences to provide a clearer view of the needs of the total individual. Continuity of service is the outstanding feature of comprehensive medicine, Dr. Evans emphasizes. Continuity not only serves the patient best, but makes possible continuity of observation and study. The natural history of certain illnesses can thus be more fully revealed, to the benefit of the patient, physician and medical student. Dr. Evans cites the example of Cornell University Medical College as an indication of new trends in medical education. Senior medical students at Cornell spend a half year in a continuous clinical clerkship encompassing all major departments of medicine. During this time the students take care of the same group of patients in the clinic, the hospital or the home, as the need arises, providing a continuing relationship.

THE most powerful and most generally useful radioactive material so far made available for industrial research has found a new home. Cobalt-60 went to work at Battelle Institute, Columbus, Ohio, where it is expected to be used to develop improved methods for chemical processing, inspection of metals and other materials, and the sterilization of foods and medicines. Institute scientists and engineers designed a massive five-ton structure of lead and concrete to shield safely 100 curies of the high-energy source. This amount, equivalent in radioactivity to $2,000,000 worth of radium, weighs around 1½ ounces and is about the size of the outer half of the thumb. The radiations given off by cobalt-60 are about equal in penetrating power to those of a 2,000,000-electron-volt x-ray machine, according to Battelle radiochemist, Ralph Belcher. "This tremendous energy," Belcher says, "will give us a powerful new tool for conducting studies extremely difficult or impractical before because of the lengthy periods of radiation necessary with other available radioactive materials."

THE average length of life among American wage-earners and their families reached an all-time high of sixty-eight and a half years in 1951, a gain of five years in the past decade, Metropolitan Life Insurance Company statisticians report on the basis of the experience among the Company's Industrial policy-holders. The statisticians forecast that, barring a major catastrophe, the average length of life for the American people will reach the biblical three score and ten before the end of the current decade.

*(continued on page 48)*

# The Surgeon's Mart

***When you are in need of surgical instruments or medical supplies you will find the dealers and manufacturers listed below ready to serve you. For your guidance when purchasing supplies we submit this list.***


## SPECIALISTS

**NEEDLES** and pins . . . **NEEDLES** and pins—
**COCHRANE** has the Sharps and the Thins!

Particularly the "hard-to-get" needles and sizes of Lane's, Bunnel's, Grieshaber, Kalt Corneal, Abscission, Adson Scalp, Adson Brain, Milliner #8—with no extra premium price tags attached.

**COCHRANE**
*PHYSICIANS' SUPPLIES, INC.*
133 E. 58th St., N. Y. 22—PLaza 3-5533 (4, 5)

## RADIUM RADON

SEEDS — APPLICATORS

THE RADIUM EMANATION CORP.
GRAYBAR BUILDING, NEW YORK 17, N. Y.
MURRAY HILL 3-8636

PRESSO® — All Cotton Elastic Bdgs.
PRESSO®-L — Rubber Reinforced Cotton Elastic Bandages
PRESSO®-LASTIK — Elastic Surgical Adhesive Tape Flesh Colored
CONTURA® — Medicated Gauze Bandages Non-Hardening
PRESSOPLAST® — Elastic Adhesive Bandages Cut Edge—Feather Edge
CRURICAST — Zinc Gelatine Bandages (Unna's Type)
HARD-COATE® — Plaster of Paris Bandages Splints

*Order from your dealer today:*

Send for authoritative reprints on the treatments of Varicosities and accompanying complications

**MEDICAL FABRICS CO., INC.**
10 Mill Street * Paterson, N. J.

## METACARPAL and THUMB FRACTURES

NEW TREATMENT NOW AVAILABLE

GOLDBERG METACARPAL SPLINT. A closed method of treating all types of Metacarpal Fractures including the Thumb.

*Write for Literature and Reprints.*

AMERICAN ORTHOPEDIC APPLIANCES
P. O. Box 31 SPRINGFIELD, MASS.

## Colostomy and Ileostomy Patients

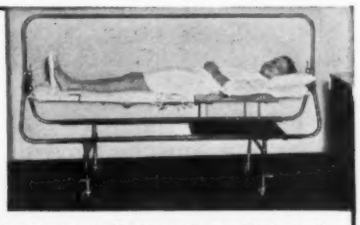
find MORE convenience and LONGER service with

**PERRY APPLIANCES**

large variety of models and sizes CUPS, BAGS, PROTECTORS and IRRIGATORS, MANY FEATURES found ONLY in PERRY appliances. Write for folders showing complete line.

**MURLE PERRY**
3803 East Lake Street
Minneapolis 6, Minn.

## STRYKER TURNING FRAME



A new development in the treatment of immobilized patients. While held firmly between two frames, the largest patient can be easily turned by the smallest nurse. Quickly elevated from either end. Easily transportable from hospital to ambulance. Essential for any modern hospital.

**ORTHOPEDIC FRAME COMPANY**
Kalamazoo, Michigan

**FOR YOUR PROTECTION**

TRADE MARK OF QUALITY

SOLE MANUFACTURER OF
**COLLISON**
SURGICAL PRODUCTS

**SURGICALLY KLEAN**
3616 FALLS ROAD
BALTIMORE 11, MARYLAND
TELEPHONE
HOpkins 4575

*The Surgical Engineering Company, Inc.*

# Outside the Ivory Tower

(*continued from page 46*)

DRUNKEN drivers face swift confirmation of their condition by police through a new test for alcohol in the blood which was described by two Cleveland chemists at the American Chemical Society's 122nd national meeting. An individual test can be run in twenty-five minutes and as many as twenty-four persons have been checked within two hours, according to Dr. Irving Sunshine and Robert Nenad, who said any large metropolitan police laboratory concerned with "a surge of alcohol determinations after a weekend of activity would benefit from a rapid test that could be done 'en masse'." The chemists are associated with the Institute of Pathology of Western Reserve University, University Hospital of Cleveland, and the Cuyahoga County (Ohio) coroner's laboratory. The new test also may save the lives of many sick and injured persons, said Dr. Sunshine, who presented the report. Hurried diagnoses in a hospital might classify as "intoxicated" a patient who actually is suffering from an injury or disease. Knowledge of the true alcohol level in the blood will reveal cases in which the patient is not drunk but suffering from some other affliction.

ASSOCIATED Hospital Service, New York's Blue Cross Plan, cooperating with three member hospitals and three visiting nursing services in the Greater New York area, will provide visiting nursing service to a limited number of members upon their discharge as hospital patients, Louis H. Pink, chairman of the AHS board of directors, announced. The cooperating hospitals are Lenox Hill Hospital, The Brooklyn Hospital and New Rochelle Hospital. The cooperating nursing agencies are the Visiting Nurse Association of New York, the Visiting Nurse Association of Brooklyn and the Visiting Nurse Association of New Rochelle. The project will be conducted on an experimental basis for a two-year period. According to Mr. Pink, the experiment, prompted by rising hospital costs, will be undertaken primarily to determine whether or not the provision of visiting nursing service for Blue Cross members might reduce the average length of time spent in the hospital. If Blue Cross members who were hospitalized patients in the Greater New York area during 1951 had left the hospital on an average of one day sooner, the over-all saving to AHS would have amounted to more than $5,000,000, he says.

Data accumulated during the two-year period will be studied to determine the feasibility of including visiting nursing service among Blue Cross benefits and to define the circumstances under which it may be provided. Patients for the experiment will be selected from private rooms, semi-private accommodations and wards on the basis of their need for nursing service after hospitalization. The need will be determined by the patient's physician, the hospital administrator, the nursing agency and a nursing "coordinator" of the project. Nursing service will be provided according to the policies of the cooperating nursing agencies. "The three cooperating hospitals will simply extend their services to include visiting nursing service for selected patients," Mr. Pink declares. "As the experiment has been planned we should acquire information on a wide variety of conditions and diagnoses which will indicate what regulations New York's Blue Cross Plan will need to make if visiting nursing service is to be included as a contract benefit."

EMPLOYEES with a total of more than 10,000 years of service to Parke, Davis & Company were honored at a dinner. Some 320 veteran employes with twenty-five years or more of service in the Detroit headquarters of the firm received gold watches from President Harry J. Loynd. Special tribute was paid to the twenty-three men and women who have been associated with the world-wide pharmaceutical firm for forty years or more. Homer C. Fritsch, executive vice president, who himself is in his 37th year with Parke, Davis, introduced this group.

Three of the group have worked for Parke, Davis more than a half-century. The longest service record of all is held by John J. Hannan, who joined the company October 25, 1900, and still is actively employed. The others are George S. Sherman, whose service dates from May 14, 1901, and Dr. Frank O. Taylor, who joined the company March 10, 1902. Longest service record of a woman is forty-six years, held by Emma L. Lipke, who has been continuously employed by Parke, Davis since December 10, 1906.

(*continued on page 50*)

# The Surgeon's Mart

*When you are in need of surgical instruments or medical supplies you will find the dealers and manufacturers listed below ready to serve you. For your guidance when purchasing supplies we submit this list.*

**STRYKER DERMATOME**

New convenience in skin grafting! Cuts grafts in 2⅜" strips, any thickness, any length, from any part of body. Works most efficiently with Stryker Skin Tape. Sewing not ordinarily required. Motor and dermatome can be autoclaved as a unit. Quickly converted to bone saw.

**ORTHOPEDIC FRAME COMPANY**
Kalamazoo, Michigan

Close-Up of Eye Taken with New Exakta "VX" Camera.

New Model with Pre-Set Diaphragm Control

**35-mm. EXAKTA "VX"**

Single Lens Reflex Camera for Parallax-Free Surgical Photography

The Exakta, with both regular and telephoto lenses, is widely used in hospitals for operation room photography. Its unique through-the-lens viewing system assures absolutely correct "on the subject" photographs during every step of the operation. In addition, this world famous medical camera is regularly used for preliminary, developmental, and end result pictures of patients; for copying X-Rays; and for making color transparencies of injuries and lesions —a necessity for recording and lecturing.

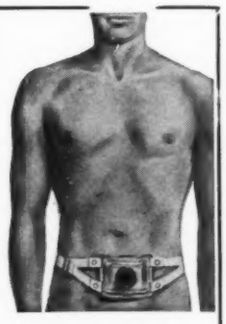
| | |
|---|---|
| With f2.8 Zeiss Tessar "T" Coated Lens with Pre-Set Diaphragm | $269.50 |
| Penta-Prism Eye Level Reflex Viewfinder | 50.00 |
| Extension Tube Set | 24.00 |
| Microscope Adapter | 29.50 |

Write Dept. 500 for free descriptive booklet "E" on camera and accessories and brochure on close-up technique with the Exakta.

**EXAKTA CAMERA CO., 46 W. 29th St., N. Y. 1, N. Y.**
Exclusive Sales and Service Organization in the U.S.A. for Ihagee Camera Works, Germany

**NEW COMFORT NOW!**
**Deodorizing**
**Colostomy Protector**

Designed for patient's comfort. Deordorizes; saves embarrassment. No bag or cumbersome apparatus. Simple design; easy to keep clean. Refer patients or write for free descriptive folder and price list to:



**ETNA Appliance & Equipment Co., Inc., Etna, N. Y.**

**ORTHOPEDIC'S UNIVERSAL BONE DRILL Has Many Features**

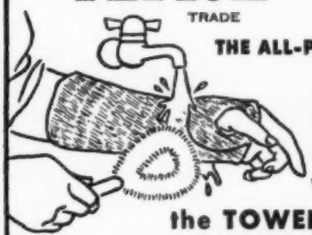
- Two-to-one gear ratio for accurate drilling.
- Smooth surfaces—nothing to catch on gloves.
- Cannulated shaft for Kirschner wires, Steinman pins, long shank drills, screw driver bits. .035" to ¼" capacity.

Send for 124 page catalog

**ORTHOPEDIC EQUIPMENT COMPANY**
300 Ft. Wayne St., Bourbon, Indiana

**AIRE-CAST**
TRADE MARK

**THE ALL-PLASTIC CAST BANDAGE**



- It's light, strong, porous
- It's X-ray transparent
- It's unaffected by water

*Write for technics and prices*

**the TOWER COMPANY, Inc.**
P. O. BOX 3181 · SEATTLE, WASHINGTON

THE GREEN JOURNAL

A Teaching Journal
on
Post Graduate Medicine

*Price:* $12.00 U.S.A.
$13.00 Canada
$15.00 Foreign

The American Journal of Medicine, Inc.
49 West 45th St. New York 36, N. Y.

**YOUNG'S DILATORS**

Extremely useful in the treatment and prevention of contracted anus (particularly following hemorrhoidectomy), as an aid in perineal dissection and repair following delivery.

**Sold only on prescription—at your surgical dealer or ethical pharmacy**

Adult Set sizes 1, 1½, 2, 3—for stretching anal sphincter.
Children's Set sizes 0, 1, 1½, 2—for preventing contracted anus.
Size 3 and 4 drilled to take towel clamps—for use in perineal repair.

***Write for Literature and Reprints***

**F. E. YOUNG & CO.** 460 East 75th Street Chicago 19, Illinois

# Outside the Ivory Tower

(*continued from page 48*)

SALES of pharmaceutical products in the United States climbed from $157,000,000 in 1939 to an estimated $1,100,000,000 in 1952. Antibiotics, particularly penicillin, set the pace in this almost unbelievable surge, and broad-spectrum antibiotics still provide a continued profit potential in the future. But many manufacturers are shifting emphasis to the production and sale of specialty drugs.

Such product diversification is necessary, Arthur F. Gormley, vice president in charge of sales for Schenley Laboratories, Inc., declared recently, if the smaller manufacturer is to survive and prosper in tomorrow's highly competitive market. He predicted that 1953 will see the increasing development, production and sale of specialties in these five major categories: (1) geriatrics, (2) cardiovascular disorders, (3) nutrition, (4) mental disturbances and (5) arthritic and rheumatoid ailments.

"Hormones are already one of the industry's top-selling items," Gormley said, "and there is every reason to believe that geriatric products offer just as promising a field for further development. With 12,000,000 American citizens sixty-five years of age or over, and with 20,000,000 predicted by 1980, a huge market already exists. It is reasonable to assume that drugs aimed at preventing or curing the chronic diseases of old age will rank among the industry's foremost sales items."

THE First World Congress on Fertility and Sterility will be held on May 25–31, 1953, at the Henry Hudson Hotel in New York City. This Congress is sponsored by the International Fertility Association with the cooperation of the American Society for the Study of Sterility. Twenty-three scientific sessions are to be held, which will embrace the entire field of fertility and sterility, including sessions dealing with socio-economic factors, psychosomatic aspects and artificial insemination. The sessions will be conducted in English, French and Spanish, with the use of earphones and simultaneous translations, as in the United Nations meetings.

In addition to the scientific sessions there will be medical round table discussions, question and answer periods, scientific exhibits and motion pictures. It is anticipated that 1,800 scientists from fifty-one countries will attend the Congress, making it the world's largest medical meeting devoted to problems of reproduction. This Congress will facilitate the exchange of ideas and information among doctors from the various countries, dealing with the very latest findings in fertility studies. Since seats at the Congress will be at a premium, it is suggested that those who plan to attend write as soon as possible to the Chairman of the Local Arrangements Committee, 1160 Fifth Avenue, New York 29, New York, for advance registration.

FINISHED plans for the erection of an additional new research building to cost $2,000,000 were approved by the board of directors of G. D. Searle & Co., Chicago manufacturer of ethical pharmaceuticals, at its regular quarterly meeting. Construction will begin at once, to be completed by next August or September. The unit represents Searle's sixth new post-war building and its second fully equipped new research laboratory.

DAVIS & GECK, INC., manufacturers of sutures and surgical specialties, recently released the twenty-first edition of its Surgical Film Library catalog for distribution to surgeons, hospitals, nurses training schools, medical schools, medical societies and other accredited medical and surgical groups. The films are loaned without charge. The current catalog of the Surgical Film Library lists some 137 subjects, many of which are Cine Clinic films from previous Clinical Congresses of The American College of Surgeons.

To meet the demands for the films, Davis & Geck has made fifteen to twenty-five prints of each subject. More than a million feet of D & G film are now clicking their way through projectors at meetings of the surgical and allied professions. This company, a unit of American Cyanamid Company, also maintains a branch library in Canada with adequate copies of films for distribution through central headquarters in Toronto. The fourteenth edition of the Canadian film catalog has also been released for distribution. Request for catalogs, both domestic and Canadian, should be addressed to Surgical Film Library, Davis & Geck, 57 Willoughby Street, Brooklyn 1, New York.

(*continued on page 52*)

# The Surgeon's Mart

*When you are in need of surgical instruments or medical supplies you will find the dealers and manufacturers listed below ready to serve you. For your guidance when purchasing supplies we submit this list.*


**dura-sharp**

**VIM**

**stainless steel needles**

will keep their keen cutting edge even after many sterilizations — the chrome-plated hub fits all U. S. standard syringes. Always specify **VIM.**

MacGregor Instrument Company Needham 92, Massachusetts

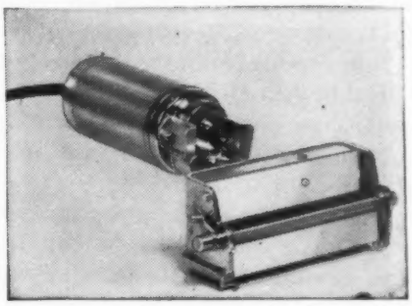
*Headquarters for*

**BONE SCREWS**

Concave cross-slot with or without pilot point.

*Zimmer*

MANUFACTURING CO., WARSAW, IND.



*The New*

**PADGETT - HOOD**

**ELECTRO DERMATOME**

Accurate and uniform grafts of any width up to FOUR inches and to any length. A single Surgeon using but one hand can operate this simple instrument without assistance

*Write to us today for detailed information*

**KANSAS CITY ASSEMBLAGE CO.**

816 Locust Street Kansas City, Mo

**GOODWIN BONE CLAMP**

Everything for the INDUSTRIAL and ORTHOPEDIC Surgeon

Complete New Organization specializing in SERVICE and QUALITY

*Write for details and Prices*

**SIEBRANDT FRACTURE EQUIPMENT INC.**

1012-14 LOCUST

KANSAS CITY 6 MISSOURI

Disintegrating

**ALESEN T-TUBE**

***For Safer Gastrectomy***

Reduces hazard of duodenal stump disruption. Smoother post-operative convalescence. Disintegrates and discharged in 5 to 7 days post-operatively. Contains barium sulfate for x-ray purposes. Available at your Surgical Supply House.

*Write for Literature*

**SEAL-INS LABORATORIES**

2857 East 11th Street, Los Angeles 23, Calif.

*Azochloramid®*

Brand of CHLOROAZODIN U.S.P.

*An Effective Anti-Bacterial Agent in*

**TRAUMATIC WOUNDS • DIABETIC GANGRENE**

**INFECTED ULCERS • DRAINING SINUSES**

*W&T Pharmaceutical Division*

**AZOCHLORAMID IN TRIACETIN 1:500**

For moistening dressings and packings

**AZOCHLORAMID SALINE MIXTURE**

To prepare isotonic saline solution 1:3300 for irrigation, hot soaks, compresses, etc.

Non-toxic to tissue — Virtually non-irritating. Non-staining — Odorless — Strongly deodorizing PA-11

1935 ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION 1952

W&T WALLACE & TIERNAN PRODUCTS, INC., Belleville 9, N. J., U.S.A.

# Outside the Ivory Tower

(*continued from page 50*)

A RARE collection of thirty-eight editions of the famous "Rosengarten" obstetrical books of the sixteenth century is now on display at the Yale Medical Library. The collection is owned by Dr. Alfred M. Hellman, noted New York physician and scholar, who has loaned the books for the Yale exhibition which is now open to the public. One of the great publications in medical history occurred in 1513 when the German physician, Eucharius Roesslin, issued his obstetrical textbook, which is commonly referred to as "Rosengarten." Written in the vernacular instead of Latin, the book was popular among the common people who knew little Latin, and had enormous influence on the practices of midwives and surgeons of that time.

The Yale exhibition contains thirty-eight different editions of "Rosengarten," or of books that were obviously based on the work of the German doctor, even to the point of plagiarism. Several of these are in English and French in addition to German and one, published in 1612 by Jacques Guillemeau, has the cheerful title of "The Happy Delivery of Women." Two of the thirty-eight books were published by the author's son, also named Eucharius, whose Latin translation of "Rosengarten" was largely used for translation into French, English, Dutch, Italian and other languages.

Roesslin's great contribution to medicine was his fight against ignorance and superstition concerning childbirth. He criticized midwives whose careless methods brought on unnecessary deaths of newborn infants, and bluntly labeled such deaths as murder for which the guilt ones deserved to be buried alive or "broken on the wheel."

THE executive office of the Pan-Pacific Surgical Association announces that the Sixth Pan-Pacific Surgical Congress will be held in Honolulu in November, 1954. Included in the scientific program will be sessions in all divisions of surgery and related fields, with papers presented by topflight surgeons from the Pacific Area countries. For further information concerning the Sixth Congress or membership in the Association, doctors may write to the Pan-Pacific Surgical Association, Suite 7, Young Building, Honolulu, Hawaii.

THE cornerstone for the new Henry W. and Albert A. Berg Institute for Experimental Physiology, Surgery and Pathology at the New York University-Bellevue Medical Center was laid, with appropriate ceremonies, on December 3, 1952. As a part of the ceremony, a check for $500,000 for construction of the Institute was presented to Winthrop Rockefeller, Chairman of the Medical Center Board of Trustees, by a representative of the estate of the late Dr. Albert A. Berg.

Dr. Berg, specialist in the field of surgery who died on July 1, 1950, provided the $500,000 in his will for construction of the institute, named for him and his elder brother, Dr. Henry W. Berg, and also provided a fund of well over $1,000,000 for its maintenance. Dr. Henry Berg, who was an authority on infectious diseases and internal medicine, died on December 21, 1938.

TWO new research grants totaling $3,000 were announced by Dr. William P. Boger, Medical Director of Sharp & Dohme, Incorporated. Principal investigator of one grant made to Tulane University, New Orleans, Louisiana, is Dr. C. G. Collins, Professor and Chairman of the Department of Obstetrics and Gynecology. This grant of $1,200 was awarded for a period of one year. Dr. Collins will conduct clinical investigation of Dirnate, Benemid and pancreatic dornase. The Cedars of Lebanon Hospital, Los Angeles, California, is the recipient of the other grant, which is in the amount of $1,800. Dr. Leonard M. Asher will conduct clinical research on Dirnate.

DUKE University psychiatrists now are grappling with "black magic," and "hexing spells" in particular. Dr. Vernon Kinross-Wright, Duke neuropsychiatrist, urged the Southern Medical Association to take a closer look at black magic and hexes. The majority of hex victims have serious mental illnesses, he declared, but some do not. For instance, a thirty-two year old man recently came to Duke Hospital suffering from gripping pains in his stomach. He could not eat and his thirst was excessive. He believed he was intended to die of starvation, imagining he had been "spelled" by a local magician for having cheated a partner in a business deal. He came

(*continued on page 54*)

# The Surgeon's Mart

*When you are in need of surgical instruments or medical supplies you will find the dealers and manufacturers listed below ready to serve you. For your guidance when purchasing supplies we submit this list.*


**POTENT ANESTHESIA**
**in Itching and Surface Pain**

**Via 20% Dissolved Benzocaine**

A rapid control of surface pain in many post-surgical conditions: Hemorrhoidectomies, episiotomies, amputations, removal of tape. Also to prevent pain in rectal and other examinations and instrumentation. Available "Clear" and "With Chlorophyll."

*Americaine*
TOPICAL ANESTHETIC OINTMENT

Send for FREE SAMPLES

ARNAR-STONE LABORATORIES, INC.
(Formerly Named Americaine, Inc.) Evanston, Ill.

*Jayne Bryant*
**SAFETY-CHECK BLANKET**

COMFORT *plus* SAFETY FOR ALL CONCERNED!

The *Jayne Bryant* Safety-Check Blanket provides safety, comfort and more freedom of movement for confused, agitated, excitable or violent patients, either hospitalized or in home confinement. *Write for complete information.*

*Jayne Bryant* **SAFETY-CHECK BLANKET**
7646 Vincennes Ave., Chicago 20, Ill.

***Dramatic New* SKIN PROTECTANT**
***Containing for the First Time*—SILICONES***
***Adhesive—Moisture Repellent—Inert***

To protect skin against maceration in colostomy drainage, ileostomy, cavernostomy, proctitis, diabetic gangrene, etc. Effective in many cases formerly failures under accepted therapy. Said to outmode zinc oxide ointment, aluminum pastes, calamine, etc.

ARNAR-STONE LABORATORIES, INC.
1316-A Sherman Ave. Evanston, Ill.

*As described in Jl. Inv. Derm., 17:125 (Sept. 1951)

**FREE** *Send for Sample and Literature*

**SILICOTE**
SILICONE OINTMENT

**FOR COLOSTOMY IRRIGATION**
**A Triple Purpose Shield**

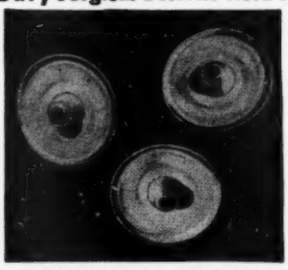
It is a positive aid in controlling the offensiveness of irrigation; allowing the patient to irrigate in the accepted manner, using a catheter of choice and inserting to desired depth.

The cover which is placed over the top of shield as soon as the catheter is withdrawn conceals all offensiveness and serves as a desk top Thus, the time necessary for patient to complete the irrigation can be devoted to some useful purpose, directing the patient's mind away from an unpleasant job.

Write today for the booklet "My Colostomy" and literature on the Trip e Purpose Shield.

E. T. WALLS, P.O. Box 144, Valhalla, N. Y.

**Davy Surgical Buttons Hold Retention Sutures Firmly**



Non-Slipping
Less Scar Formation
Clean—Comfortable
Made of Aluminum

•

Excellent for Mastectomy as well as Laparotomy

•

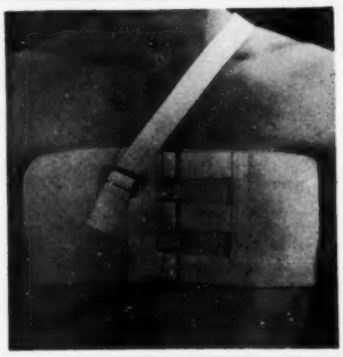
FREE SAMPLES, upon request
At Your Dealers

**AMERICAN MEDICAL SPEC. CO. INC.**
12 East 12th Street New York City

**FRACTURE EQUIPMENT**
**Traction Apparatus, Finger Splints, Head Halters, Walking Heels**

*Catalog Sent on Request through Surgical Dealers.*

℞ RAJOWALT COMPANY
WARSAW, INDIANA



***ELASTIC* RIB BELTS FOR FRACTURED RIBS**

More comfortable and efficient than adhesive strapping. Made of the finest quality elastic webbing. Order through your dealer, or order direct with this ad. Delivered. Less 10% on orders of 12.

SHIP TO:

☐ Large Adult . . . $2.75 each
☐ Adult . . . . . $2.50 each
☐ Medium Adult . $2.25 each
☐ Child . . . . . $2.00 each

**LITTLE MFG. CO., BOX 202, WADESBORO, N.C.**

# Outside the Ivory Tower

(*continued from page 52*)

to Duke because he had heard that the psychiatrists could hypnotize people and "take things off their minds," but he reserved the right to visit a "conjure man" in a nearby city before Duke doctors actually treated him. "I heard some three weeks later," Dr. Kinross-Wright said, "that the hex has been successfully removed and the patient was in good health."

This man was not a severe mental case and could not be called such, the Duke doctor declared, unless the same label were put on most of his friends and relatives who confirmed the alleged hexing. "A man who thinks he has been placed under the influence of a spell is not in a normal state," the Duke neuropsychiatrist says. "He must, of course, be highly suggestible and must also have committed some act that makes him feel guilty."

Duke doctors can only try to explain to such patients the absurdity of their beliefs. They cannot throw up their hands in despair and give up trying to understand hex victims.

A NEW synthetic drug is speeding up the time it takes to prepare patients for certain surgical operations, two Yale University anesthetists reported. The drug, succinylcholine, has been used successfully in nearly 1,000 operations since April at the Grace-New Haven Community Hospital. These were all operations in which rapid relaxing of body muscles is required.

The Yale anesthetists reported its use in abdominal, thyroid, heart and chest, and neck and head operations of many types. The drug's most marked success is in relaxing muscles of the jaw and throat so that tubes may be inserted into patients' windpipes to facilitate breathing during surgery. Analysis of more than 700 of these operations is contained in a paper read at the annual meeting of the American Society of Anesthesiologists. The paper was written by Doctors L. Jennings Hampton, Associate Professor, and David M. Little, Jr., Assistant Clinical Professor of Anesthesiology at Yale's School of Medicine. Dr. Hampton also is Director of the Department of Anesthesiology of the Grace-New Haven Community Hospital.

EACH of the nation's Governors has an invitation to name a seventy-five-year old doctor to attend the first western hemisphere conference of the World Medical Association as a guest of honor. It will be held at Richmond, Virginia, April 23 to 25, 1953. The invitations were extended by John S. Battle, Governor of the host state, to the forty-seven other Governors, asking that a senior physician from each state come to Virginia and bear witness to medical advances that have taken place within his lifetime. The seventy-five year old guests, who carried medicine to their first patients in saddlebags or by horse and buggy, will travel to Virginia by Pullman or plane. They will be greeted by Dr. Louis H. Bauer, president of the American Medical Association, and by medical leaders of Latin America.

A NEW scientific research laboratory, specially designed for the safe study of tuberculosis and other diseases caused by microorganisms, was opened by The Wm. S. Merrell Company, it was announced by Nelson M. Gampfer, president. Gampfer said tuberculosis, which kills more persons each year than all other infectious diseases combined—one every twelve minutes—will comprise the lion's share of study. He was quick to explain, however, that laboratory researchers will also study the diseases caused by bacterial organisms of pneumonia and typhoid fever, the viruses of influenza and poliomyelitis, and the sometimes fatal fungus infections.

REAR ADMIRAL Wincell McKendree Craig, (M.C.), U. S. Naval Reserve, was installed as the new president of the Association of Military Surgeons of the United States at its fifty-ninth annual meeting. He will serve for one year.

DR. LOUIS H. BAUER, President of the American Medical Association, said that the length of human life as measured for industrial workers is now sixty-eight years. At the same time it was announced that a regional meeting of The World Medical Association will be held in Richmond, Virginia, April 23 and 24, 1953. The annual World Medical Association will meet this year at the Hague in September.

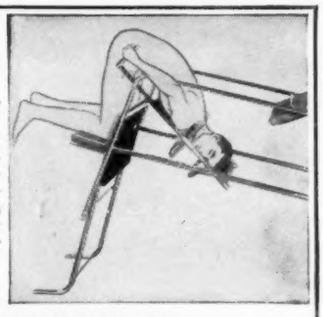
# The Surgeon's Mart

*When you are in need of surgical instruments or medical supplies you will find the dealers and manufacturers listed below ready to serve you. For your guidance when purchasing supplies we submit this list.*


***The exclusive***
**EDER-McCLURE**
EXAMINING TABLE FOR RECTAL AND ROUTINE EXAMINATIONS

Patented proctological position recommended by specialists provides comfort, yet patient cannot move during instrumentation. Priced lower, yet it is two tables in one. Converts in 30 seconds to a handsome examining table.

*Write for Descriptive Folder 108*



**EDER INSTRUMENT CO.**
2293 Clybourne St., Chicago 14, Illinois

BRAND NEW GOV'T SURPLUS
**SURGICAL INSTRUMENTS**
and Medical Supplies

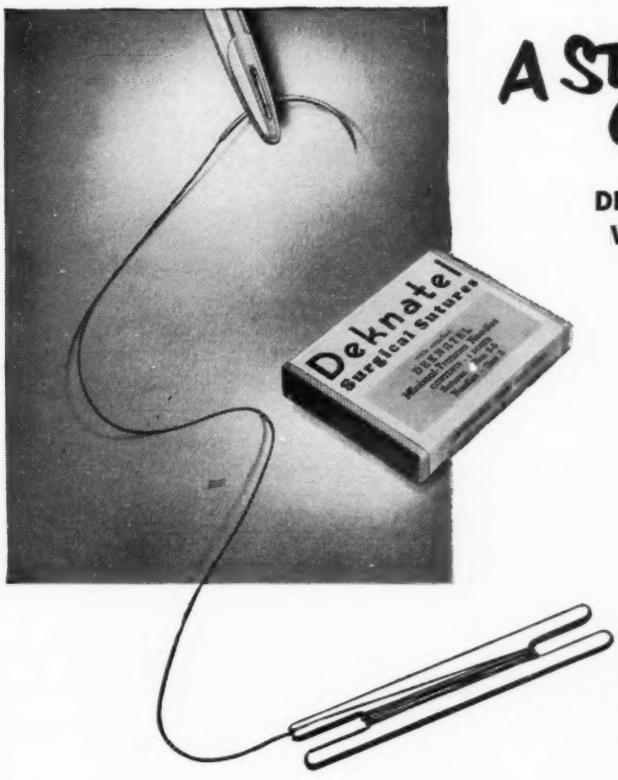
AT 50% BELOW REGULAR COSTS
all merchandise on approval
no payment unless perfectly satisfied
write for illustrated catalog L
**ARISTA SURGICAL COMPANY**
67 LEXINGTON AVE., NEW YORK 10, N. Y.

**THE PRIMER BANDAGE**
(moist,)
combined with
**THE DALZOFLEX BANDAGE**
(elastic adhesive)
SUGGESTED IN THE PRESSURE TREATMENT
OF
LEG ULCERS, VARICOSITIES
and other ailments of the extremities
where pressure is indicated

It costs no more to use the best
Catalog and price-list on request

*Medical Aids Inc.*
5 North Wabash Avenue Chicago 2, Illinois


In answering advertisements please mention The American Journal of Surgery


**A STREAMLINED COMBINATION—**

**DEKNATEL SURGICAL SUTURES WITH MINIMAL-TRAUMA NEEDLES ATTACHED**

- Suture securely and permanently anchored to needle
- Saves time of needle threading and preparation
- Needles are of tempered, polished steel with sharp points and keen cutting edges
- Deknatel Silk Sutures are moisture and serum resistant—uniformly high in tensile strength

For full details write
J. A. Deknatel and Son,
Queens Village 29, (L. I.) N. Y.

**DEKNATEL**
**Needle Sutures**

Other Deknatel Products: Surgical Silk, Nylon, Cotton—Readi-Cut Sutures—Readi-Wound Ligature-Reels—Name-on Beads

# NEW

*Davis & Geck's Spiral Wound Surgical Gut on a cylindrical reel!*

*Saves time makes easier ties*

Surgeons welcome a new convenience—D & G Spiral Wound Surgical Gut. It is wound on a cylindrical reel—comes *ready for immediate use* in ligation and suturing. Spiral winding preserves all the original strength. None is lost through kinks or bends.

Nurses, too, save time with Spiral Wound Gut. No more unwinding from an old style flat reel and rewinding onto a rubber tube, glass rod or spindle.

**For surer hand ties**

When the surgeon makes a series of hand ties, D & G Spiral Wound Surgical Gut USP unreels in his hand, free from kinks, with all its tensile strength preserved.

The gut should be gently pulled out straight immediately after removal from the tube.

**D & G foresees the surgeon's needs**

D & G Spiral Wound Gut is the latest Davis & Geck contribution to improved suturing. "Timed-absorption" surgical gut is another—this exclusive D & G method embodies accurately graded degrees of chromicizing. The suture resists digestion most strongly during the first postoperative days, when greatest strength is needed. It is absorbed more rapidly when tissues have regained their natural strength.

*Davis & Geck, Inc.*

A UNIT OF AMERICAN *Cyanamid* COMPANY

57 Willoughby Street, Brooklyn 1, N.Y.

# The American Journal of Surgery

## ADVERTISERS INDEX

*March*, 1953


Abbott Laboratories....16, 43
American Cystoscope Makers, Inc.... 11
American Hospital Supply Corp.... 4
American Medical Specialties Co., Inc.... 53
American Orthopedic Appliances.... 47
Ames Company, Inc.... 1
Arista Surgical Co.... 55
Arnar-Stone Laboratories, Inc.... 53
Austenal Laboratories, Inc.... 31
Bard-Parker Co., Inc.... 10
Bauer & Black....*Insert Facing Page* 8
Baxter Laboratories.... 4
The Berivon Company.... 14
Jayne Bryant Safety-Check Blanket Co.... 53
Chatham Pharmaceuticals, Inc.... 37
Ciba Pharmaceutical Products, Inc....*Back Cover*
Cochrane Physicians' Supplies, Inc.... 47
Cook County Grad. School of Medicine... 34
Crescent Surgical Sales Co.... 35
Cutter Laboratories.... 33
Davis & Geck, Inc....17, 56
J. A. Deknatel & Son, Inc.... 55
Dome Chemicals, Inc.... 35
Drug Publications, Inc....40–41
Duke Laboratories, Inc.... 36
Eaton Laboratories, Inc.... 9
Eder Instrument Co.... 55
Ethicon Suture Laboratories, Inc....
*Insert Facing Page* 24
Etna Appliance & Equipment Co., Inc.... 49
Exakta Camera Company.... 49
E. Fougera & Co., Inc.... 23
Gomco Surgical Mfg. Co.... 45
Kansas City Assemblage Co.... 51
Kny-Scheerer Corporation.... 22
Lederle Laboratories....*Insert Facing Page* 16
Eli Lilly & Co.... 32
Little Manufacturing Co.... 53
Macalaster Bicknell Parenteral Corp.... 36
MacGregor Instrument Co.... 51
Mead Johnson & Company....12–13
Medical Aids, Inc.... 55
Medical Fabrics Co., Inc.... 47
Merck & Co., Inc.... 26
V. Mueller & Co.... 27
N. Y. Polyclinic Medical School & Hospital. 34
Orthopedic Equipment Co.... 49
Orthopedic Frame Company....47, 49
Outside the Ivory Tower....46, 48, 50, 52, 54
Parke, Davis & Company.... 29
Murle Perry.... 47
Chas. Pfizer & Co., Inc....8, 19–20–21
George P. Pilling & Son Co.... 28
Propper Manufacturing Co., Inc.... 30
The Radium Emanation Corp.... 47
Rajowalt Company.... 53
A. H. Robins Co., Inc.... 15
Seal-Ins Laboratories, Inc.... 51
G. D. Searle & Co.... 42
Sharpe & Dohme, Inc.... 24
Siebrandt Fracture Equipment, Inc.... 51
E. R. Squibb & Sons.... 6
The Surgeon's Mart....47, 49, 51, 53, 55
The Surgical Engineering Co., Inc.... 47
The Tower Co., Inc.... 49
The Upjohn Company.... 25
Wallace & Tiernan Products, Inc.... 51
E. T. Walls.... 53
Wilmot Castle Co.... 18
Winthrop-Stearns, Inc.... 2
Wyeth, Inc.... 38
Louis Yellin, Inc.... 39
F. E. Young & Co.... 49
Zimmer Manufacturing Co.... 51

*Long-lasting relief*

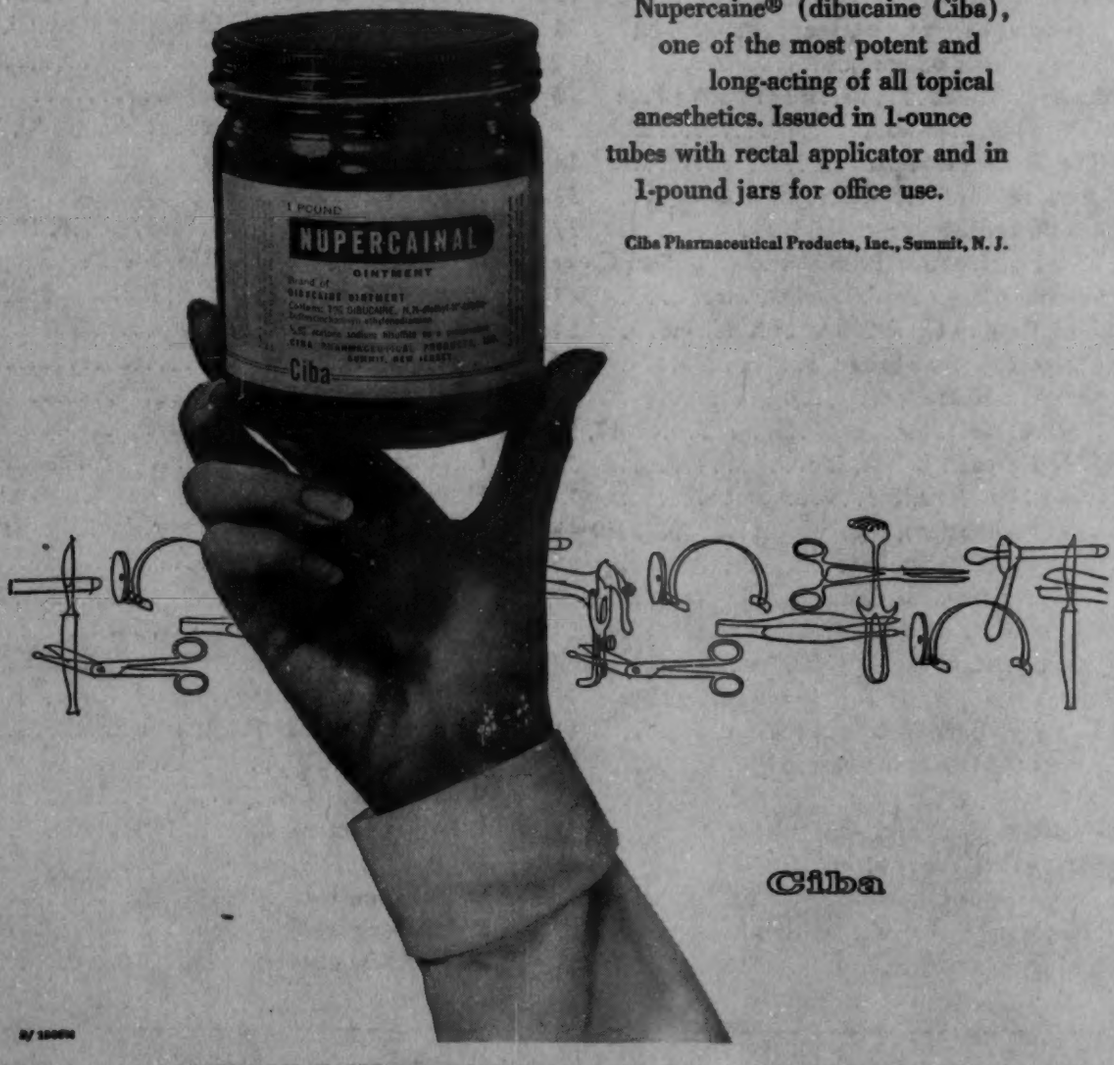
*of surface pain and itching*

*with* **Nupercainal®**

***potent . . . . . nonirritating . . . . . nonnarcotic***

**The effective and prolonged surface anesthesia provided by Nupercainal Ointment (dibucaine ointment Ciba) brings long-lasting relief from pain and itching in sunburn, hemorrhoids, abrasions, dermatoses and many other conditions. Its effectiveness is due to its 1 per cent content of Nupercaine® (dibucaine Ciba), one of the most potent and long-acting of all topical anesthetics. Issued in 1-ounce tubes with rectal applicator and in 1-pound jars for office use.**

**Ciba Pharmaceutical Products, Inc., Summit, N. J.**




**Ciba**